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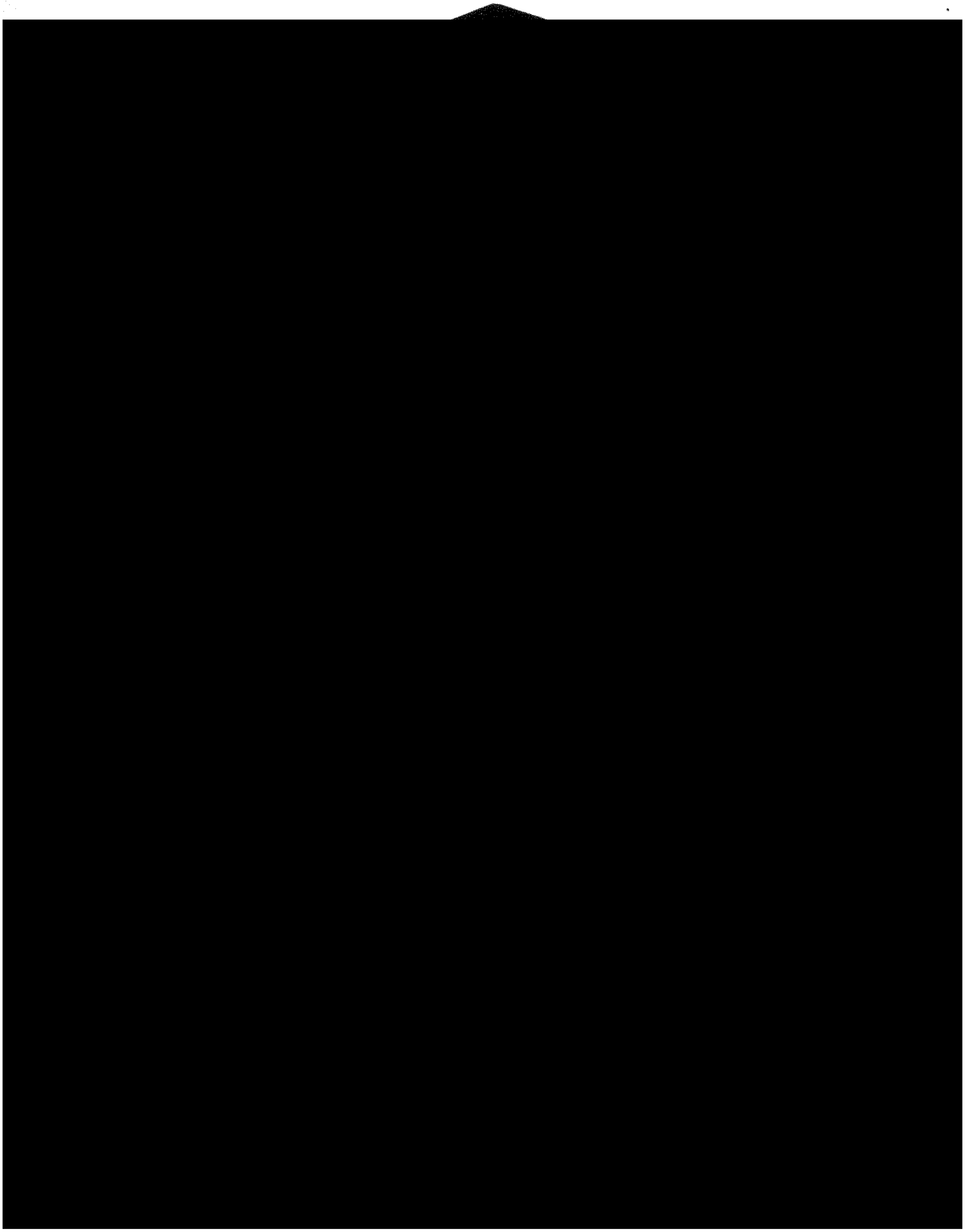

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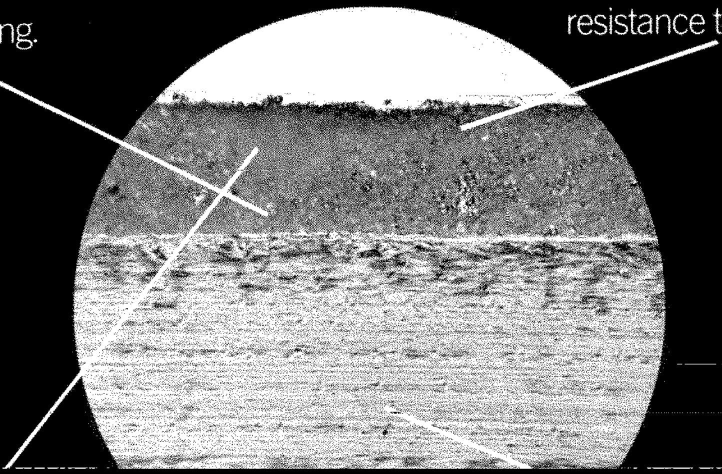

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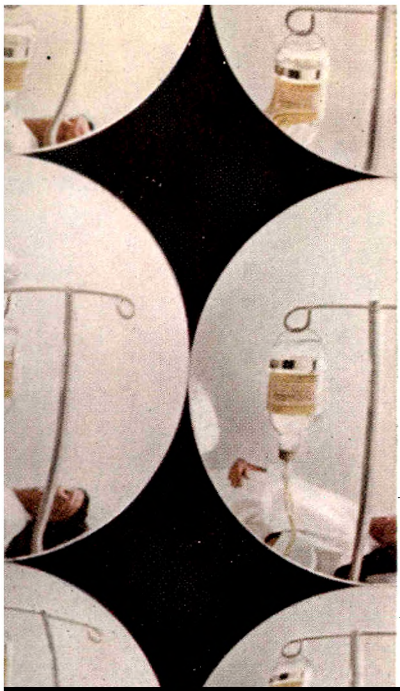

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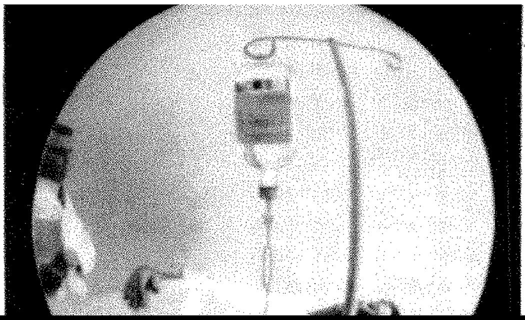

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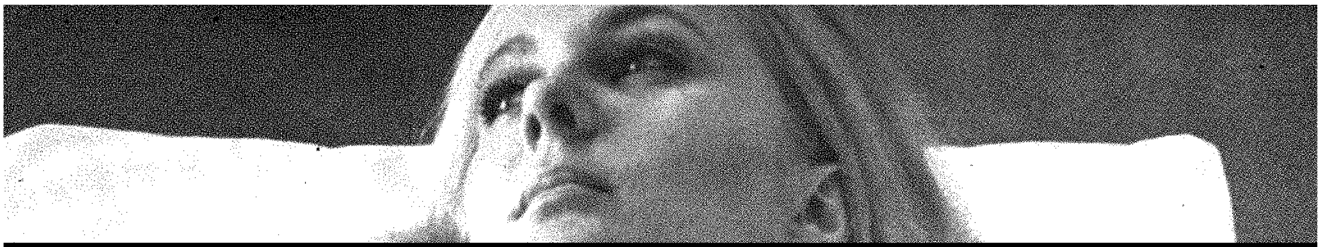

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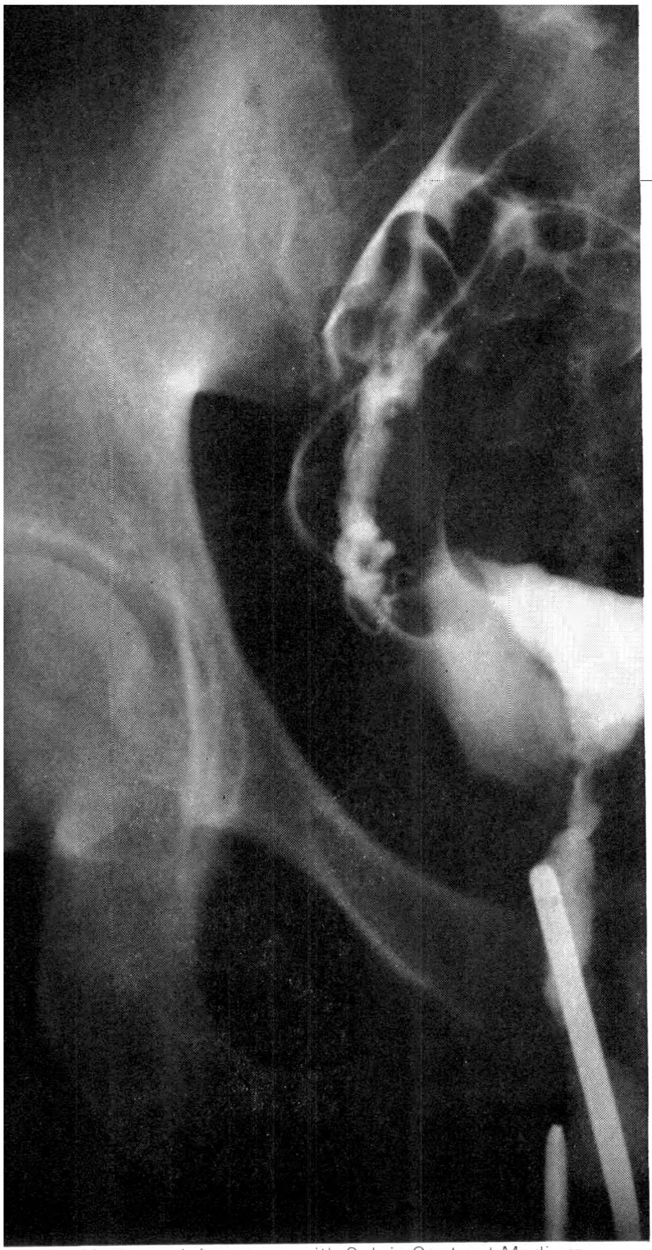

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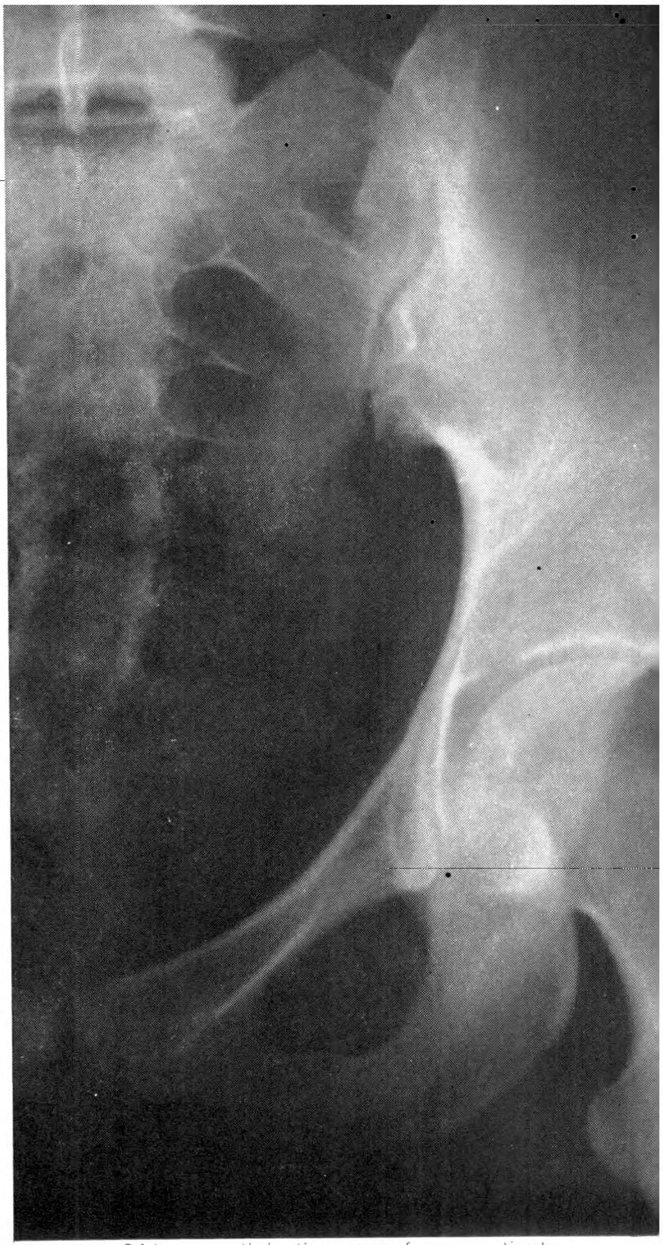

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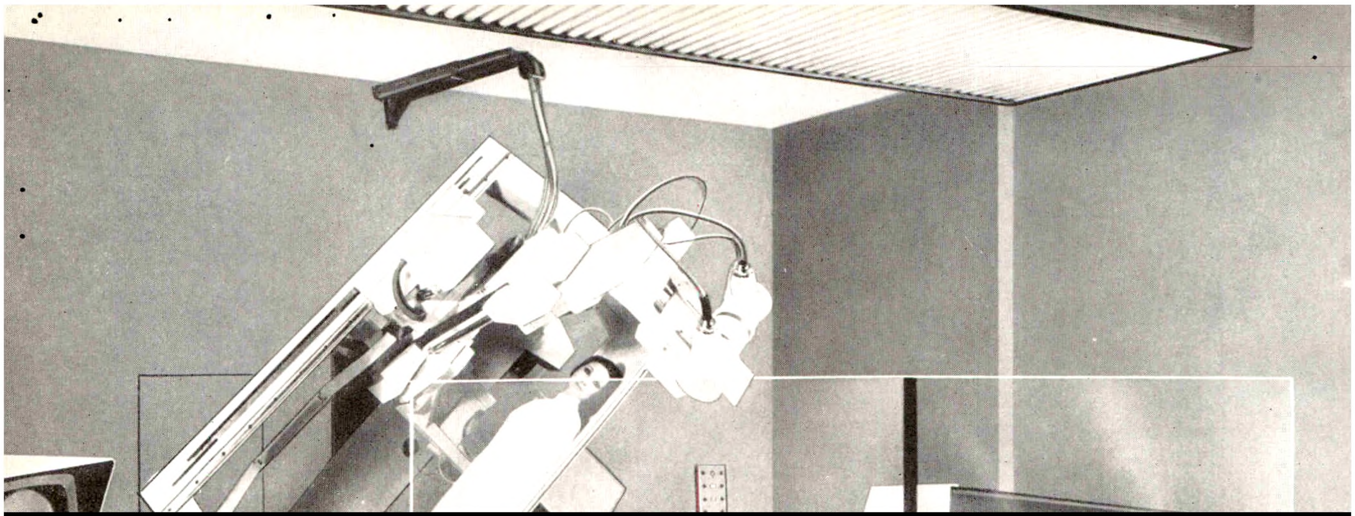

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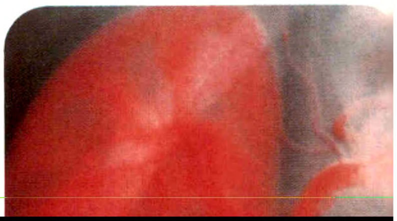

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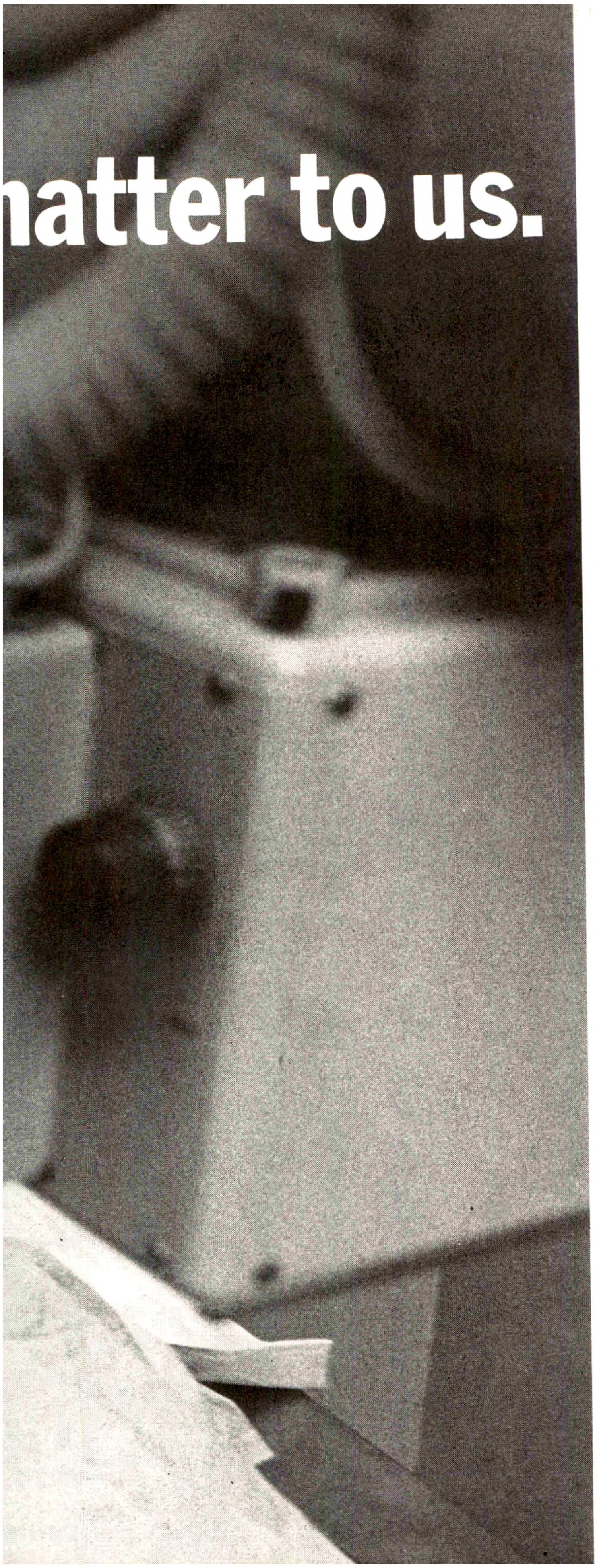

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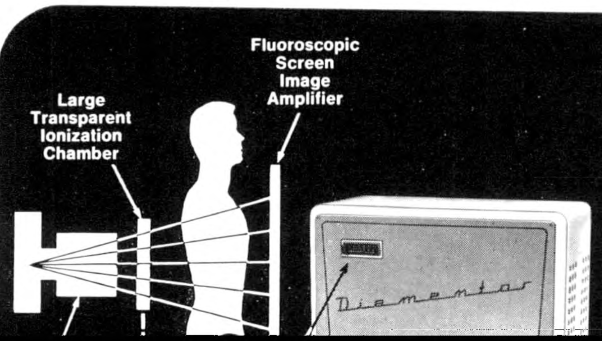
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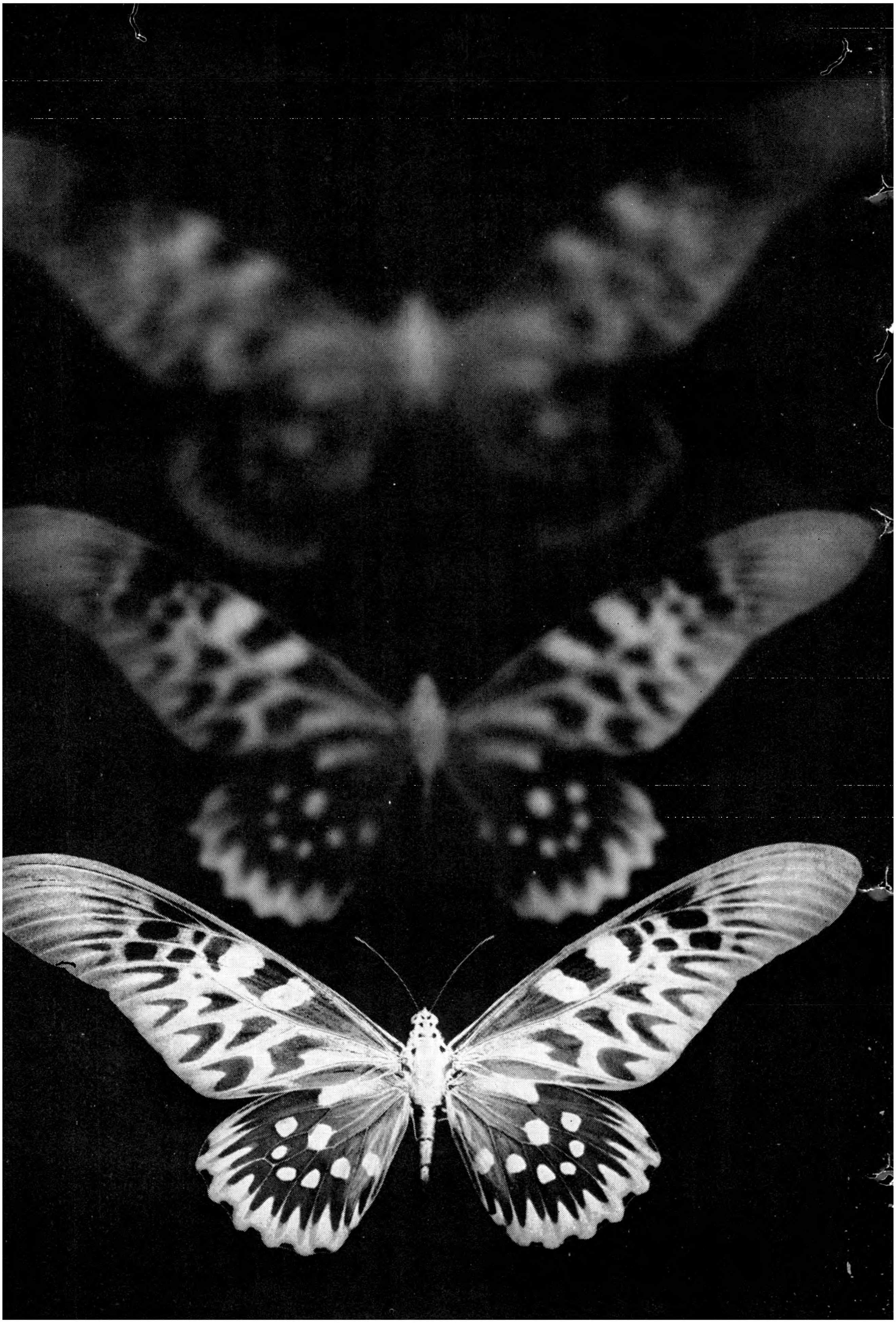

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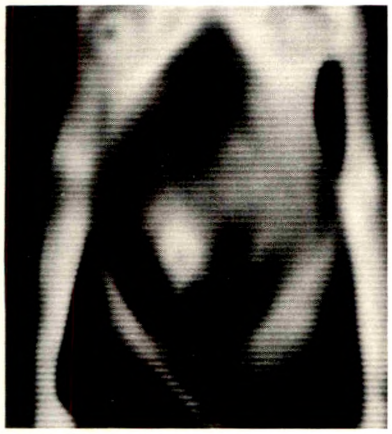

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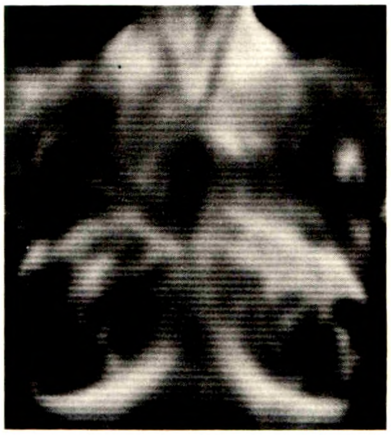

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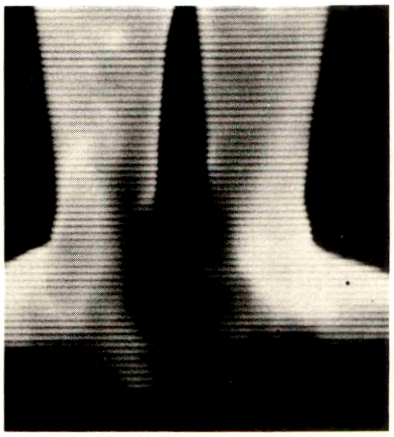

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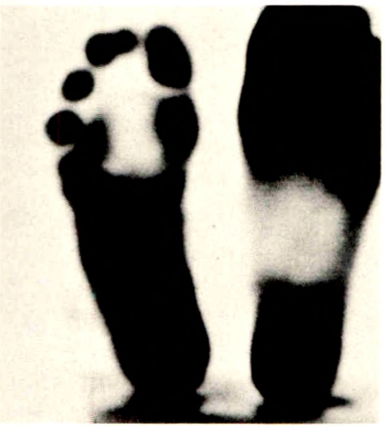

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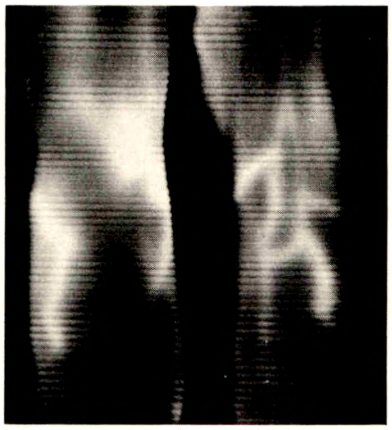

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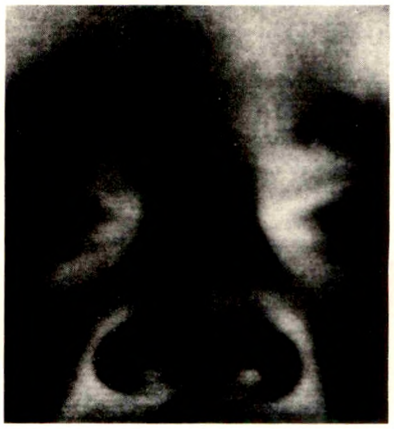

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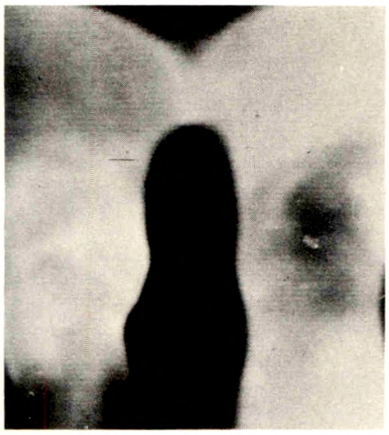

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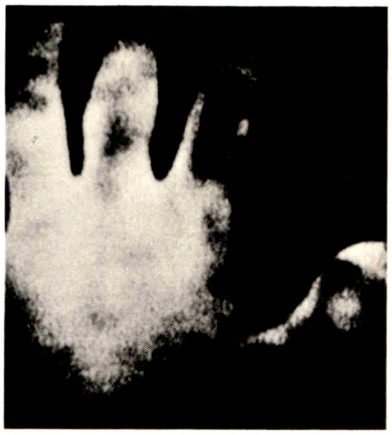

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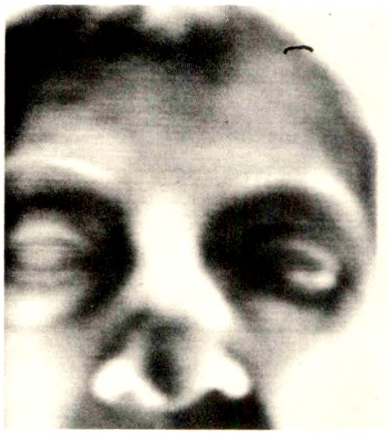

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Associated abnormalities of the surrounding bony structures, including a high arched hard palate, and an increased thickness of the vomer, nasal septum and the lateral nasal structures are often present, thereby reducing the diameters of the nasal fossa and the nasopharynx. The frequent association of other congenital anomalies has been well documented[4] and a familial incidence has been noted by some authors.[18] In the large series of Flake and Ferguson,[3] there was a twofold predominance of females over males. In patients with unilateral choanal atresia the occurrence was predominantly right-sided (over 2:1).

SYMPTOMS

Ronaldson in 1881[8] recorded the first clinical description of choanal atresia and it has never been surpassed in clarity.

Despite the classic description of bilateral choanal atresia some 89 years ago, many physicians are still unaware of the lethal potential of this uncommon anomaly of the newborn. It is not generally appreciated that the newborn infant instinctively breathes only through the nose, and that oropharyngeal breathing is an acquired function, which normally takes several days to weeks to learn. This aversion to mouth breathing has been attributed to an evolutionary need for air currents to pass over the olfactory area.[7] The anatomic cross sections of the infant skull performed by Moss[5] demonstrate that the relatively higher position of the cervical viscera during infancy impedes access of air from the mouth to the lower airway and that the nose is the best avenue for respiration. It may be that this structural basis for obligatory nasal breathing in the newborn plays a protective role against the aspiration of food. Because most neonates are obligate nasal breathers, bilateral choanal atresia usually precipitates respiratory distress immediately after birth. This anomaly, therefore, must be included in the differential diagnosis of any newborn infant suffering from respiratory distress. Failure to consider this life threatening entity, establish the correct diagnosis, and promptly institute appropriate therapy is undoubtedly responsible for an undetermined number of neonatal deaths from asphyxia annually in the U.S.A. In those infants who escape diagnosis in the first hours of life because they rapidly learn mouth-breathing, respiratory distress is usually precipitated subsequently by attempts at feeding, since the ability to simultaneously breathe and suck is seldom achieved until age 3–6 weeks.

All infants with bilateral choanal atresia develop a thick, glairy, mucoid discharge from the nostrils. The copious, leathery mucus may occasionally become blood tinged but secondary infection seldom supervenes. The external nares and upper lip may become excoriated from the constant mucus discharge from the nose. Affected patients are unable to blow the nose and the voice develops a nasal quality. Anosmia and diminished taste are present. The true diagnosis in those few patients who do not manifest neonatal respiratory distress is often delayed, and the nasal obstruction is erroneously attributed to allergy, rhinitis, deviated nasal septum or enlarged adenoids.

The recent proposal by Shaw,[11] that bilateral nasal obstruction from trivial upper respiratory infection in the small sleeping infant who does not instinctively or voluntarily breathe through his mouth is a significant factor in sudden unexpected death of infancy, is an attractive one. More than 15,000 infants under 6 months of age die annually from this perplexing problem and it constitutes the greatest cause of mortality during the first year after the neonatal period (2 to 3/1,000 live births).

NEW CLINICAL CLASSIFICATION

A new clinical classification of bilateral choanal atresia, based on the rapidity with which the newborn learns to both breathe orally and to co-ordinate mouth breathing and sucking, is proposed by the author (Table II). It is hoped that this classifica-

Table I

SUMMARY OF CASES

Case	Sex	Location of Atresia	Type (bilateral cases)	Associated Anomalies
1. B.G.H.	F	Bilateral	I (Fig. 1, *A* and *B*)	
2. D.M.	F	Bilateral	II	
3. G.J.	M	Bilateral	I	Esophageal atresia with fistula
4. R.A.L.	M	Bilateral	III (Fig. 3, *A* and *B*)	
5. A.L.	F	Left	(Fig. 5, *A* and *B*)	
6. J.M.C.	M	Right	(Fig. 4, *A* and *B*)	Omphalocele
7. B.G.D.	F	Bilateral	III	
8. J.H.	F	Left		Ventricular septal defect and congenital deformity of external right ear
9. M.H.	M	Right		
10. L.C.	F	Bilateral	II (Fig. 2, *A* and *B*)	
11. M.W.	M	Right		
12. G.W.	M	Left		
13. K.S.	F	Right		
14. L.S.J.	F	Left		Mongolism
15. J.A.	M	Bilateral	I	
16. C.D.	F	Right		
17. T.M.	F	Bilateral	I	
18. B.W.	F	Bilateral	I	
19. R.S.	F	Right		

tion, by stressing the clinical rather than the anatomic aspects of the disease, will lead to a better understanding of the various manifestations of bilateral choanal atresia and hence to an earlier diagnosis.

Type I. These infants lack the ability to rapidly learn the technique of oropharyngeal breathing and immediately develop air hunger, cyanosis and chest retraction (Cases 1, and 3). Type 1 comprises the largest group (Table 1) and these infants will die from asphyxia unless an artificial airway is immediately established. Prolonged use of such an airway is often required, but in many cases the infant can be gradually weaned from it over a period of weeks. These infants frequently cannot co-ordinate mouth breathing and sucking and attempts at feeding provoke respiratory distress. Gavage feedings are usually necessary in such infants and must be continued after oropharyngeal breathing has been mastered, until such time as the necessary co-ordination between mouth breathing and sucking develops.

Type II. This smaller group of infants learns mouth breathing more rapidly after birth than do infants of Type 1. Their respiratory distress is cyclic and less severe, necessitating only transient or intermittent use of an artificial airway (Case 2). However, even after the technique of oropharyngeal breathing has been mastered (hours to days), respiratory distress may still be provoked by attempts at feeding. The obviously ravenous infant will suckle only spasmodically because the nipple causes temporary occlusion of the oral airway. Until such infants learn to co-ordinate oral breathing and sucking (usually by the age 3–6 weeks), gavage feedings should be employed. Persistent, premature attempts to orally feed such infants prior to development of this co-ordination will result in choking, cyanotic spells and aspiration pneumonitis.

TABLE II

CLASSIFICATION OF BILATERAL CHOANAL ATRESIA

	No. of cases	Time required to master mouth breathing	Time required to develop co-ordination between oral breathing and sucking	Type of neonatal respiratory distress	Airway requirements	Need for gavage or specialized nipple feedings
Type I	5	Prolonged (weeks)	3-6 weeks usually	Immediate, severe and prolonged; usually ag-	Immediate, prolonged	Frequent

[redacted]

decongestant into each nostril will eliminate the possibility of mistakenly diagnosing nasal obstruction by edematous, inflamed nasal mucosa for choanal atresia. This is particularly important where the mother has been receiving reserpine. Nasal congestion occurs in approximately 10 per cent of such infants and when severe may occlude the nasal passages, thereby simulating bilateral choanal atresia.[6] This drug-induced nasal congestion may persist for 5 to 6 days following delivery. If desired, the thickness of the occluding septum can be determined by measuring the distance separating the opaque medium in the posterior nasal cavity from the air column in the nasopharynx. The opaque medium must be flushed from the initially examined nare before proceeding to examine the opposite nare. If prior use of an airway has been found essential to prevent respiratory distress, it should be left in place during the roentgenographic examination.

Unilateral choanal atresia is usually a benign condition with minimal symptoms, and is seldom diagnosed until later in childhood or adult life (Case 5). The chief symptoms consist of a persistent nasal obstruction and a constant discharge from the affected nostril. Unilateral atresia, however, may assume real importance in early infancy should the unobstructed nostril become occluded from rhinitis or other cause prior to mastery of the technique of mouth breathing. Under such circumstances a unilateral choanal atresia becomes functionally converted into a bilateral choanal atresia and severe respiratory distress may develop (Case 6).

TREATMENT

Since bilateral choanal atresia often presents as a neonatal emergency, immediate diagnosis and treatment are essential to prevent death from asphyxia. Failure to consider this congenital anomaly in the newborn infant with severe respiratory distress is responsible for an indeterminate number of readily preventable deaths annually. In cases of Type I bilateral choanal atresia, immediate establishment and maintenance of an adequate oropharyngeal airway is essential and lifesaving. Gavage feedings are often necessary subsequently in such infants. Corrective surgery, the definitive treatment, can be performed later as an elective procedure. In Type II infants, intermittent use of an airway and gavage feedings should be employed. Type III cases require only gavage feedings during the neonatal period and these may be discontinued when the afflicted infant learns to co-ordinate oral breathing and swallowing. Type IV cases require no therapy initially.

The definitive treatment in all types of bilateral bony choanal atresia is surgical resection of the septa. Surgical resection of the obstructing bone via the transpalatal approach is most commonly employed and this operation can be successfully performed during the neonatal period.[1] The posterior choanal diameter doubles during the first year of life and for this reason surgery is often delayed until after 6 months of age, at which time it is technically simpler. More recently McGovern[4] has described the use of a large holed rubber nipple which is strapped in the infant's mouth as a temporary means of treatment. This ingenious, yet simple device was invented by the concerned parents of a child born with bilateral choanal atresia. By employing the McGovern apparatus, definitive surgery may be safely delayed for several months, when it becomes technically simpler because of the increased size of the operative area. A modification of the McGovern nipple has been successfully employed in several of the more recent cases at our hospital (Case 7).

In cases of bilateral membranous atresia, transnasal puncture of the occluding membranes with a curved hemostat, followed by insertion of hollow tubing through each nostril into the nasopharynx, can be readily performed during the neonatal period. Should stenosis occur after this procedure, it can be treated by periodic dilatations.

In patients with unilateral atresia (Case 5) surgical correction of the anomaly can

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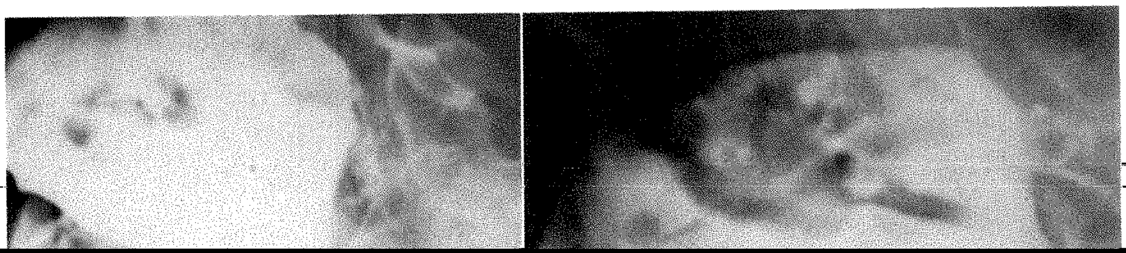

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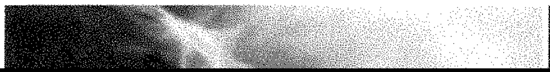

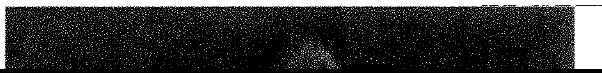

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mentioned series, where unilateral atresia occurred twice as frequently on the right side.[3]

Because of the author's dissatisfaction with the anatomic classification of bilateral choanal atresia which offers no assistance to the clinician, a new classification of bilateral choanal atresia has been devised and is proposed (Table II). This is a clinical classification, based on the rapidity with which the neonate learns to mouth-breathe and to co-ordinate oral breathing with sucking. It is hoped that this classification, as opposed to the traditional anatomic classification, will orient the clinician towards the varying clinical features of bilateral choanal atresia. Hopefully this approach will lead to earlier diagnosis and institution of appropriate treatment.

The diagnosis of bilateral choanal atresia is established clinically by failure to pass a rubber catheter through either nostril into the nasopharynx. Roentgenologic confirmation can be achieved by injecting an opaque medium such as oily dionosil into each nasal cavity. The initial treatment in symptomatic infants consists of establishing an airway and employing specialized feeding techniques. The recent introduction of the McGovern nipple has provided an ingenious alternative form of temporary treatment and allows definitive surgery to be delayed until it is technically simpler. This simple technique solves both the respiratory and feeding problems and obviates the need for an endotracheal tube or tracheotomy in most cases.

Unilateral atresia is usually a benign condition and the diagnosis is frequently not established until later in life, when the affected individual presents with the complaint of unilateral chronic nasal obstruction and discharge. Only rarely does unilateral choanal atresia produce a life threatening situation, but this can occur if the uninvolved nostril becomes occluded by rhinitis or other cause before the infant learns oral breathing (Case 6). Except in these rare instances, the treatment of unilateral choanal atresia is elective.

SUMMARY

1. Posterior choanal atresia is an uncommon condition, but it is not as rare as the scant radiologic literature would indicate.

2. The findings in 19 cases of unilateral and bilateral choanal atresia are presented, with the emphasis on symptoms, diagnosis, and treatment. It is stressed that the newborn infant is an obligate nasal breather and that oral breathing is an acquired ability.

3. A new clinical classification of bilateral choanal atresia, based on the ease with which the neonate learns both to breathe orally and to co-ordinate mouth breathing and sucking, is proposed as a replacement for the traditional anatomic classification.

4. Bilateral choanal atresia must be ruled out in any neonate with respiratory distress. Failure to quickly establish the diagnosis and institute immediate therapy may result in preventable death from asphyxia.

5. Unilateral choanal atresia is usually a benign condition and elective therapy suffices.

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The author wishes to express thanks to the various radiologists and otorhinolaryngologists in various Twin City Hospitals who contributed cases and encouragement.

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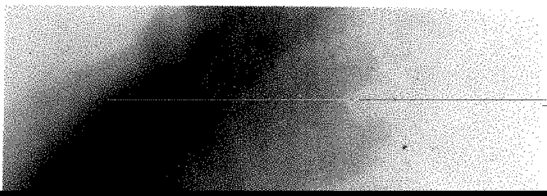
ANTERIOR TRACHEAL INDENTATION IN INFANCY AND EARLY CHILDHOOD: NORMAL OR ABNORMAL?*

By LEONARD E. SWISCHUK, M.D.
GALVESTON, TEXAS

THE hazards of assessing tracheal contour in the infant and young child are well known, and there is no question that normal tracheal buckling and tortuosity can be misinterpreted as being representative of underlying pathology. However,



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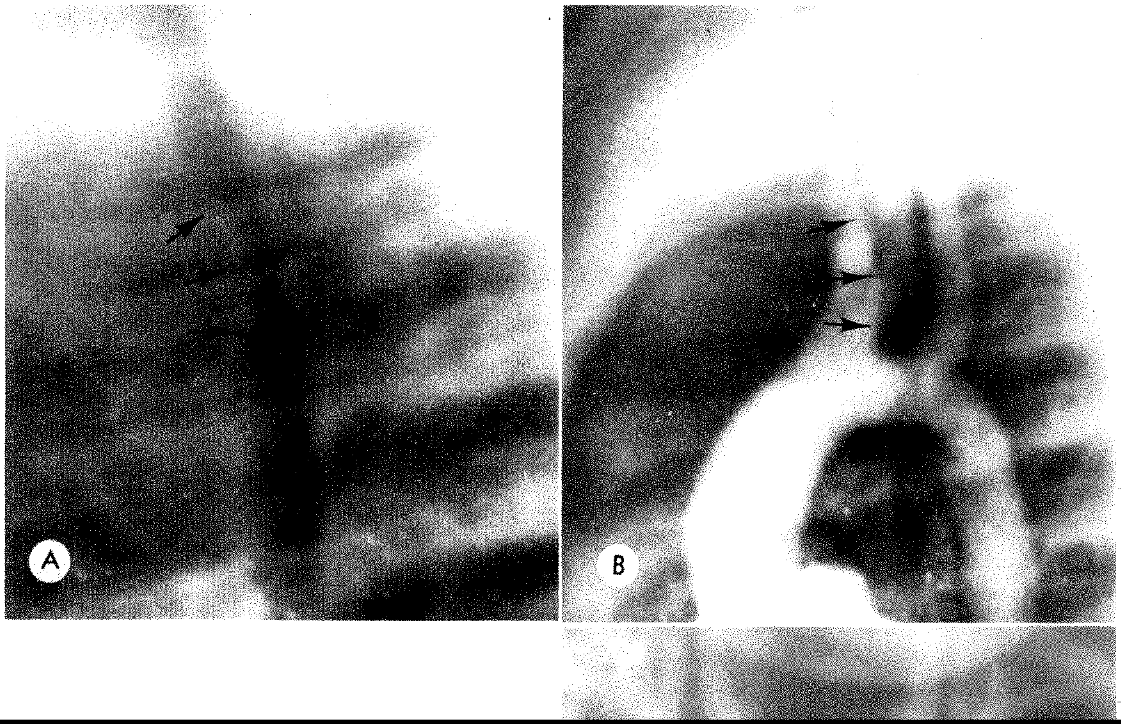

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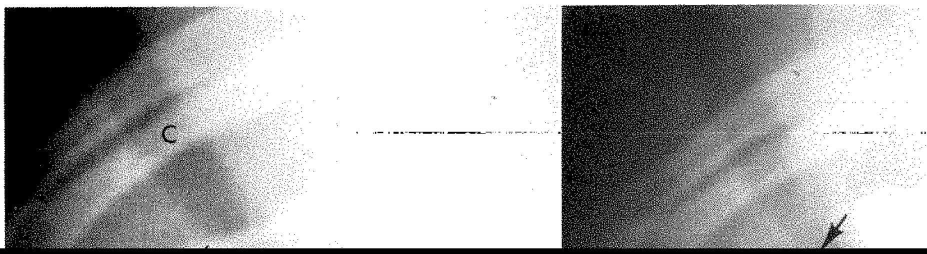
even lesser degrees of indentation were often symptomatic.

In explaining this seeming discrepancy, it is postulated that other factors, such as tracheal cartilage softness or underdevelopment (tracheomalacia), tracheal smallness, tracheal inflammation with mucosal edema, and tautness or exact position of the offending vessel are also, or perhaps even mainly, involved.[1,7] In other words, the innominate artery, tracking across the trachea, is probably more a factor of predisposition than one of primary cause of obstruction. If this were not the case, many more infants with symptomatic tracheal indentation would be encountered.

In considering the natural course and eventual fate of the indentation, it became apparent that some changes did occur, for anterior tracheal indentation is much less common in older children. In fact, it is unusual for anterior tracheal indentation to



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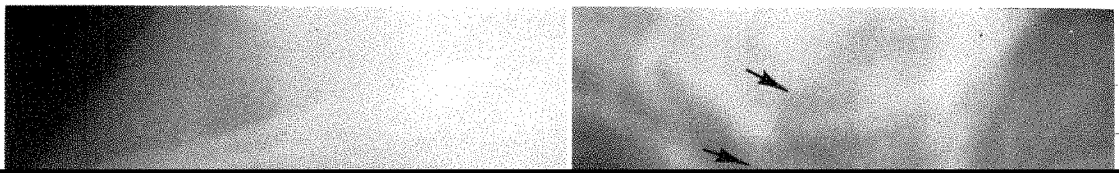

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contents (*i.e.*, the mediastinal structures occupy a relatively greater space than in adults), it is believed that the superior mediastinum is overcrowded.[1,7] Consequently, all of the structures are "compressed" and, as a result, the likelihood of the innominate artery indenting the trachea

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be entirely asymptomatic (Fig. 4, *A–C*). In addition, infants can be shown to have a definitely anomalous origin and course of the innominate artery and yet have no indentation or signs of obstruction (Fig. 5, *A–C*).

RESPIRATORY OBSTRUCTION IN THE NEWBORN AND EXCESS PULMONARY FLUID*

By DAVID P. CORBETT, M.D., *and* JAMES E. WASHINGTON, M.D.
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RESPIRATORY obstruction characteristically leads to obstructive emphysema or atelectasis. When the obstruction develops *in utero*, while the lung is still full of fluid, the roentgen appearance in the neonatal period will be different. Roentgenograms made early in life will show an increased volume of the lobe or lung distal to the obstruction, with an apparent mass density due to the retained pulmonary fluid. This entity has been reported previously[5] in cases of extrinsic occlusion of the left main stem bronchus by a small bronchogenic cyst and in atresia of the bronchus leading to an accessory left upper lobe.

The present report concerns 2 newborn infants who presented with excess pulmonary fluid and increased volume of lung due to intrauterine obstruction to the airway. One infant subsequently developed the classic features of congenital lobar emphysema. The other had stenosis of the right main stem bronchus associated with anomalous origin of the ~~left pulmonary~~ artery.

REPORT OF CASES

CASE I. T. L., a 5 day-~~old white female, was~~ admitted because of respiratory distress. She was the product of a full term uneventful pregnancy and delivery. Her birth weight was 9 lb., 6 oz. One hour following birth there was evidence of circumoral cyanosis and she was placed in an isolette. A chest roentgenogram (Fig. 1) showed increased volume of the right lung with uniform opacification except at the apex and base. No air bronchogram was evident. There was displacement of the heart and mediastinal structures to the left.

The patient showed slow but gradual clinical improvement and roentgenograms made 2 days later (Fig. 2) showed a persistent increase in volume of the right hemithorax with gradual clearing of the pulmonary density. There was now a rather diffuse reticular pattern throughout the mid portion of the right lung which was believed to be due to absorption of fluid via pulmonary lymphatics. There was continued displacement of the heart and mediastinal structures to the left. The patient continued to improve and was discharged home. Chest roentgenograms obtained 2 weeks later (Fig. 3) showed complete clearing of the previously noted parenchymal density. There was now overinflation of the right middle lobe with some associated compression of the upper and lower lobes and persistent displacement of the heart and mediastinal structures to the left. The patient was re-admitted to the hospital at this time.

Positive physical findings were limited to the

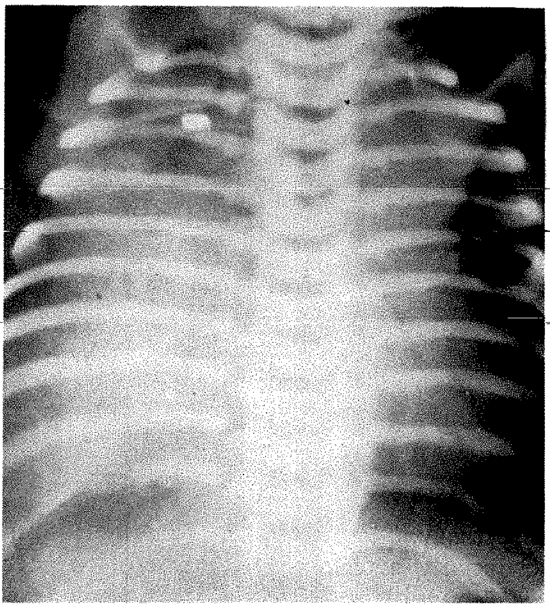

FIG. 1. Case I. Age 1 hour. Frontal roentgenogram of the chest shows increased volume of the right hemithorax with displacement of the heart and mediastinum to the left. There is uniform opacification except at the apex and base. Note the absence of an air bronchogram.

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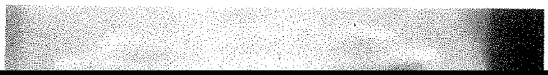

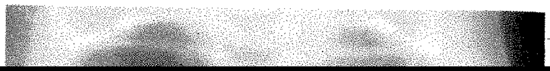

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DISCUSSION

It has been well established that the

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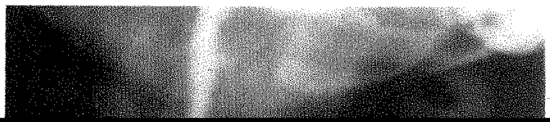

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LOCALIZED ROENTGENOGRAPHIC PATTERN OF HYALINE MEMBRANE DISEASE*

EVIDENCE THAT THE UPPER LOBES OF HUMAN LUNG MATURE EARLIER THAN THE LOWER LOBES

By RONALD C. ABLOW, M.D., *and* MARCELLO M. ORZALESI, M.D.
NEW HAVEN, CONNECTICUT

THE alveolar surface of the lungs is normally lined by a thin layer of surface-active material known as "surfactant." This material, presumably a phospholipid, lowers the surface tension of the air-filled interface and facilitates the expansion as well as the stability of the alveoli. Destruction, inactivation, or lack of surfactant results in low compliance of the lungs and a marked tendency toward atelectasis.[14]

Observations in lambs[4,6] and in rabbits[8] have shown that the upper lobes of the lungs develop their surface tension properties earlier in gestation than the lower lobes. It is possible that this pattern of surfactant maturation is similar for the human lung, since it has been shown that there are certain similarities in the developmental processes of all mammalian lungs.[7]

Using the direct magnification technique[1] we observed a roentgenologic pattern in 3 infants with the clinical picture of hyaline membrane disease (HMD) which suggests that the human lung follows a sequence of maturation of surfactant similar to that found in fetal lambs and rabbits. These 3 cases are summarily reported.

CLINICAL AND ROENTGENOLOGIC MATERIAL

The relevant information regarding the state at birth and the clinical course of the 3 premature infants is summarized in Table I. All babies exhibited, soon after birth, the clinical sign of HMD including tachypnea (respiratory rates between 70 and 110), chest retractions, grunting (Silverman score between 4 and 8), and cyanosis in room air. They all required increased oxygen concentrations (50 to 80 per cent) and infusions of sodium bicarbonate to maintain adequate arterial oxygen tensions and pH. During the acute phase of the disease the arterial pO_2 ranged between 44 and 55 mm. Hg and the pCO_2 ranged between 48 and 58 mm. Hg. The duration of the symptoms varied between 48 hours and 4 days, and all 3 infants recovered. The pertinent chest roentgenographic findings in the 3 patients at various ages during the course of their illness are shown in Figures 1–3. All 3 cases, during the course of their disease, showed at one time or another a fine reticulogranular pattern in the lower lung fields, which was not present in the upper portions of the lungs.

DISCUSSION

The 3 infants had a birth weight between 2.4 and 2.7 kg. and an estimated gestational age of 35–37 weeks. There was no clinical evidence of aspiration at birth and the clinical picture in each case was consistent with the diagnosis of hyaline membrane disease.

The fine reticulogranular pattern present in the lower lung fields in all 3 cases was identical to the generalized pattern seen in more severe cases of HMD.

The precise etiology of HMD is not known; however, it has been shown by various authors that in HMD (or the idiopathic respiratory distress syndrome) there is a deficiency or inactivation of surfactant that could account for most of the typical

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Study supported in part by United States Public Health Service Grant No. 5-MO1-RR-00125-06.

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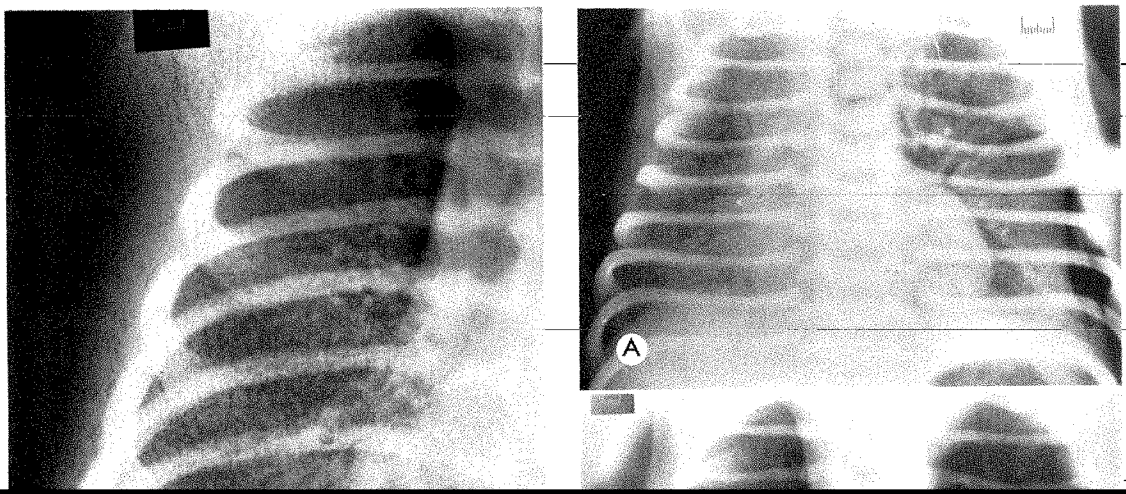

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asphyxia, while animals at term are very resistant to asphyxia and very rarely develop respiratory distress. On the basis of the above mentioned clinical and laboratory observations, it has been suggested that HMD is related to two main factors: (1) the degree of maturation of the surfactant system of the lungs and the amount of surfactant "reservoir" present at birth; and (2) the degree of asphyxia or other functional derangements taking place in the perinatal period.[2,5,9–11,13]

Howatt *et al.*[6] have shown that the upper lobes of the lungs of fetal lambs develop their suface properties earlier in gestation than the lower lobes. The normal gestational period for lambs is 140 days; before 120 days gestation the lungs of fetal lambs were found to collapse fully after inflation; between 120 and 130 days gestation, the upper lobes of the lungs retained more air than the lower lobes, the difference being most marked at 125 days. Morphologic, biochemical and surface tension studies of the lungs of these animals showed that maturation of the upper lobes preceded the lower lobes during this transitional period.[4]

Similar findings have also been obtained in rabbits by Orzalesi and Motoyama.[8] In fetal rabbits of 27 days gestation or less, both upper and lower lobes of the lungs collapsed after inflation; at 27½–28 days gestation only the upper lobes retained air normally, and at 29 days or more all lobes of the lungs remained inflated with air.

In the human fetus, surfactant is first detectable in minute amounts as early as the 23rd week of gestation[12] and at 28 weeks is usually present in amounts sufficient for normal lung expansion and stability.[3] The production of surfactant continues during gestation so that in full term infants a "reservoir" of surfactant is probably present at birth.

It is therefore conceivable that when the infant is not very immature and when there is mild perinatal stress, HMD will be induced only in those areas of the lungs where surfactant is present in lesser amounts. It is also possible that in mild cases with generalized involvement initially, recovery will take place more rapidly in those areas of the lungs which are more mature.

This was probably the case in our 3 patients who were only slightly premature and had little evidence of perinatal distress at birth. In these patients the upper lobes were either spared (Case I and II) or cleared more rapidly (Case III), indicating that the pattern of maturation of the surfactant system of the lungs in the human infant is probably the same as in other animals, *i.e.*, the upper lobes mature faster than the lower lobes.

These findings would not have been appreciated if a conventional roentgenographic technique had been used instead of direct magnification roentgenography which provides better resolution[1] (Fig. 2, *A* and *B*).

SUMMARY

Roentgenograms by the direct magnification technique were obtained in 3 premature infants with the clinical picture of hyaline membrane disease.

In 2 infants the typical roentgenologic features of the disease were limited to the lower portions of the lung fields, while in the third patient a diffuse pattern was seen, but the upper lobes cleared faster than the lower lobes.

It is postulated that the maturation of the lungs in the human fetus follows a pattern similar to that described in lambs and rabbits, *i.e.*, the upper lobes mature earlier than the lower lobes of the lungs.

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PULMONARY SLING*

ROENTGEN OBSERVATIONS

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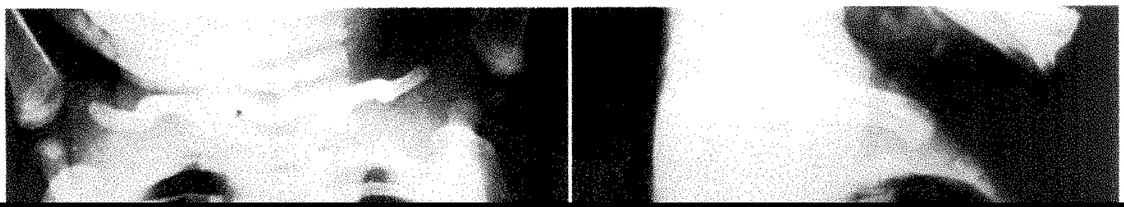

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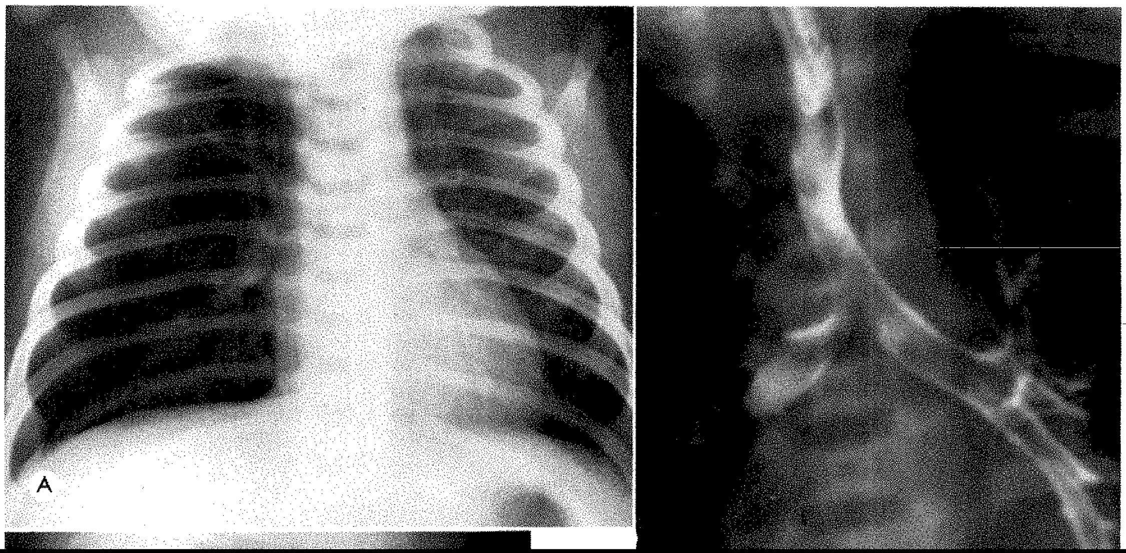

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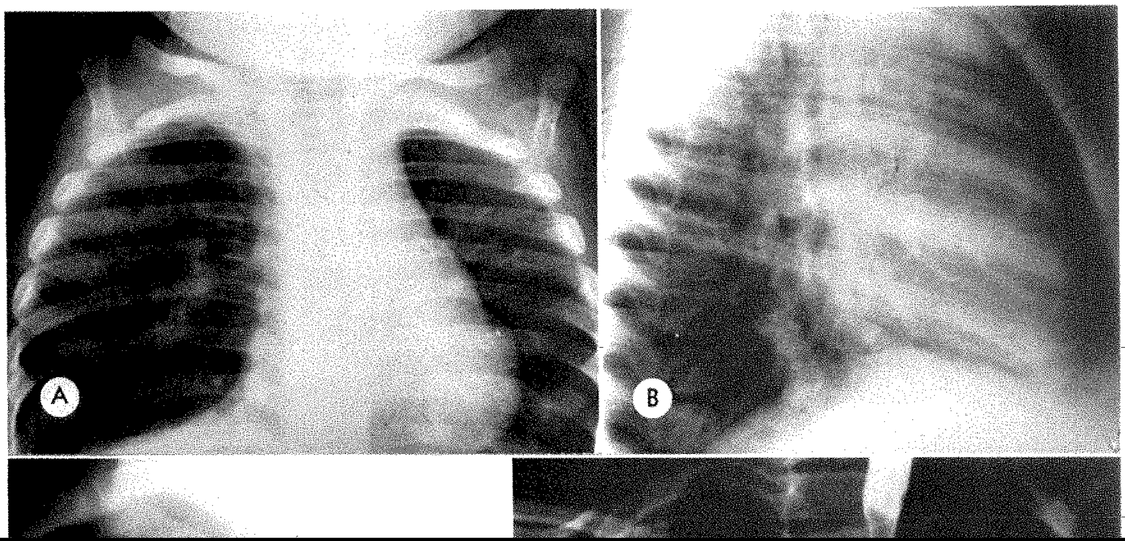
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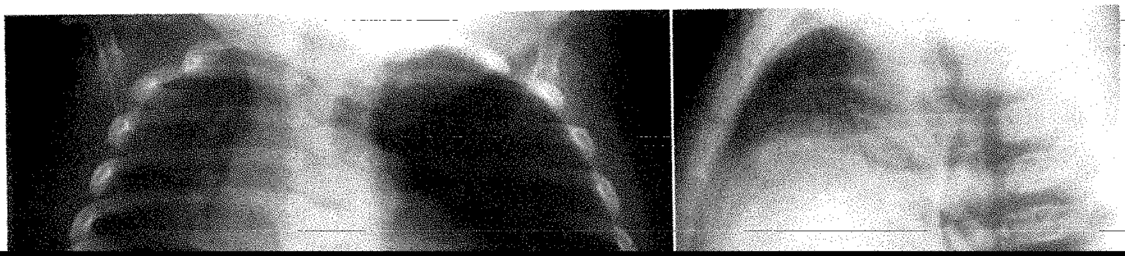

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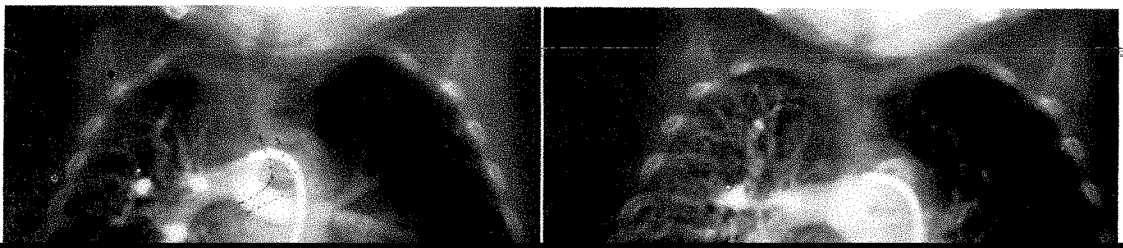

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THYMIC ENLARGEMENT AND PLEURAL EFFUSION: AN UNUSUAL ROENTGENOGRAPHIC COMPLEX IN CHILDHOOD LEUKEMIA*

By FRANK MAINZER, M.D., *and* HOOSHANG TAYBI, M.D.
SAN FRANCISCO, CALIFORNIA

ALTHOUGH leukemia is the most com- [redacted]

REPORT OF CASES

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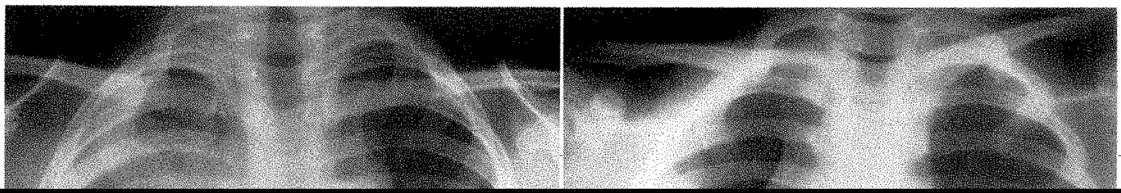

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thymic tissue. Metcalf[10,11] has further implicated the thymus in lymphatic leukemia by the demonstration of a lymphocytosis-stimulating factor in mice and human plasma. In mice, the only source of this substance was the thymus.

The frequency of thymic enlargement or

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and McCreadie.[4] Their patients, however, as well as our 5 cases, had been treated initially with steroids. Adrenocorticosteroid therapy causes atrophy of the thymus, which was well demonstrated by Caffey and di Liberti[2] in chest roentgenograms of human infants.

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ANGIOCARDIOGRAPHY IN HUGE HYPERTROPHY OF THE THYMUS

By A. CASTELLANOS, M.D.,* R. PEREIRAS, M.D.,† *and* O. GARCIA, M.D.‡
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HUGE hypertrophy of the thymic gland in infants is not uncommon[1,6–10,18,20,25,27] and at times a differential diagnosis from mediastinal tumors, intrapericardial tumors, pericardial effusion, and cardiomegaly, is necessary. Therefore, knowledge of the angiocardiographic findings in patients in whom an unusual or bizarre enlargement of this gland is present, appears to be of value.

The medical literature on this subject is very sparse.[5,6] There are other methods to differentiate hypertrophy of the thymus from cardiomegaly and mediastinal tumors,[3,4,11,13,16,18,20–29] but in this article we wish to present our angiocardiographic experience.

MATERIAL AND METHOD

A series of 150 cases was studied in the X-Ray Department and in the Pediatric Cardiology Department in the period of 1938 to 1959. The patients' ages ranged from 4 days to 6 years, although the vast majority were from 2 to 9 months of age. Plain telecardiograms were made in the anteroposterior and lateral projection, and the tube to target distance was 6 feet. Artificial anterior pneumomediastinum or pneumomediastinography (P.M.) was performed according to our own technique when necessary.[9] In most of the cases angiocardiographic studies were also done by injecting opaque medium through one of the veins of the left elbow or through the external left jugular vein. A No. 18 or 16 Lindeman cannula was introduced in the vein and injection of the contrast material (50 or 75 per cent hypaque) was carried out manually (1½ cc./kg.). When a catheter was placed into the left subclavian vein, the injection was made with the aid of an automatic injector, and using a Fairchild magazine or the Shönander apparatus the films were exposed at the rate of 3 to 5 per second. Anteroposterior or both anteroposterior and lateral projections were employed. Two cases had to be taken to the operating room: 1 with an intrapericardial tumor and 1 with a bronchogenic cyst of the superior mediastinum.

RESULTS

We have found that cases of huge hypertrophy of the thymus gland do not show any significant displacement of the right and left cardiac cavities, pulmonary arteries, pulmonary veins, or aorta. The superior vena cava and the innominate vein appear to be normal as well. Several demonstrative cases will be presented which will substantiate this statement.

REPORT OF CASES

CASE I. Figure 1 is a plain roentgenogram in the anteroposterior projection of an infant 3 months old; a bilateral hypertrophy of the thymic lobes is seen. Figures 2 and 3 are the dextro-angiocardiogram and the levo-angiocardiogram, respectively, of this patient. As may be noted, they are normal in spite of the 0.71 ratio of the cardiothymic silhouette to the transverse diameter of the base of the thorax.

CASE II. A 4 month old infant was referred to the Radiology Department because of moderate suprasternal and intercostal retractions. Episodes of transient stridor, cough and slight cyanosis were noted. The electrocardiogram was normal and no cardiac murmur was heard. Figure 4 demonstrates a high bilateral enlarge-

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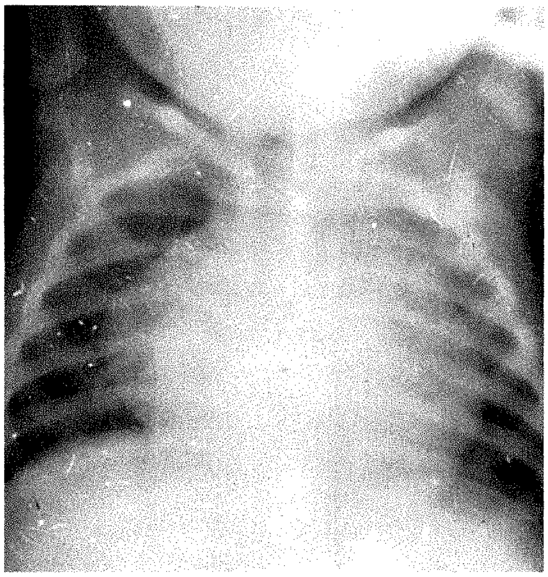


Fig. 1. Case 1. Plain roentgenogram in anteroposterior projection. Huge bilateral hypertrophy of both right and left lobes of the thymus gland.

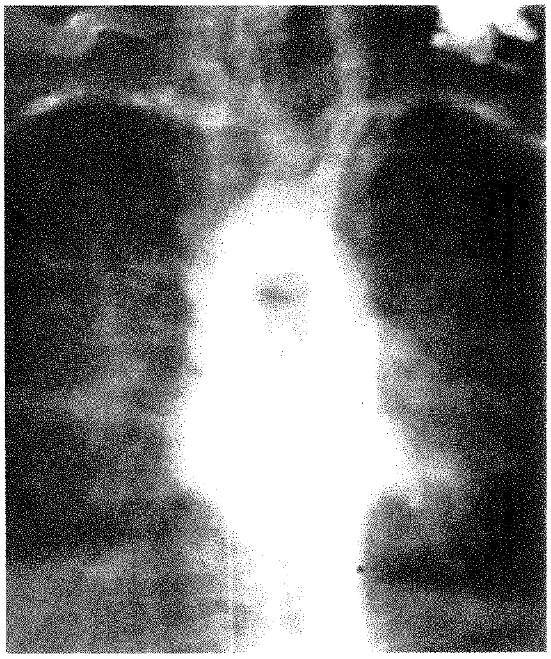
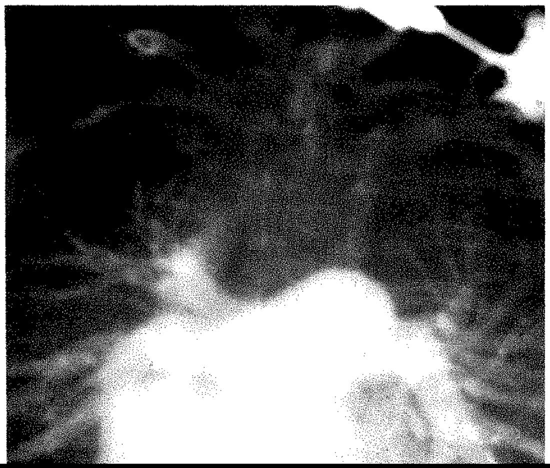
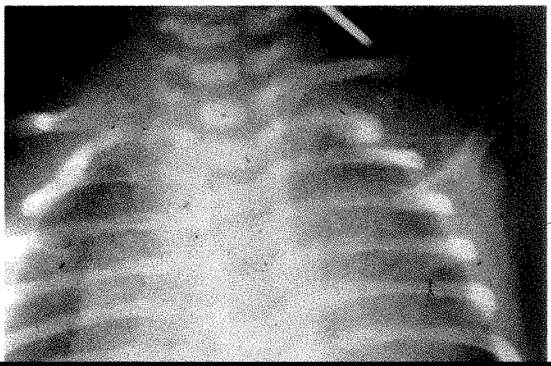


Fig. 3. Case 1. Normal levo-angiocardiogram. Exposure made in the left ventricular systole.

ment of the thymus. Figure 5 is the dextro-angiocardiogram in the anteroposterior projection. The outflow tract of the right ventricle appears to be slightly enlarged, but that should be considered a normal variant. Figure 6 is the levo-angiocardiogram which is also normal. In this case, as in the above mentioned case, the cardiac chambers and great vessels are surrounded by thymic tissue. Figure 7 is the artificial anterior pneumomediastinum in the antero-





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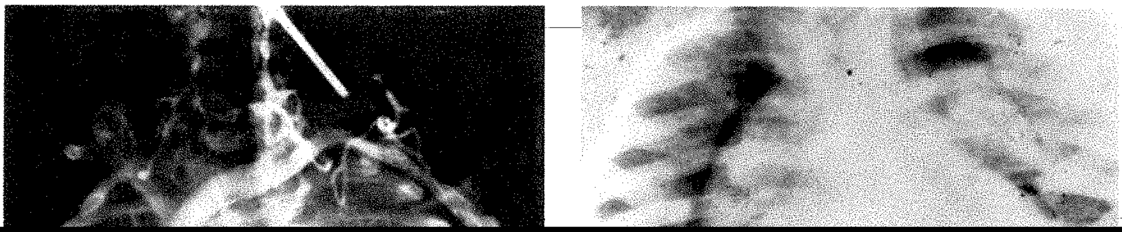

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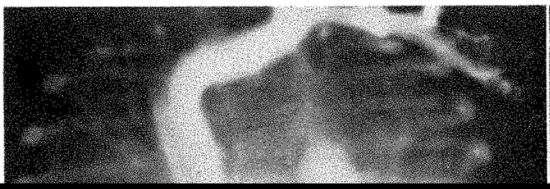

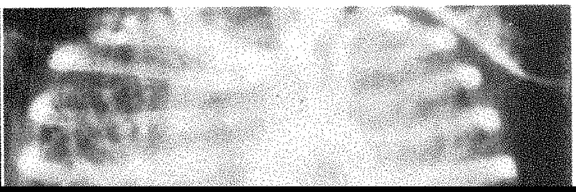

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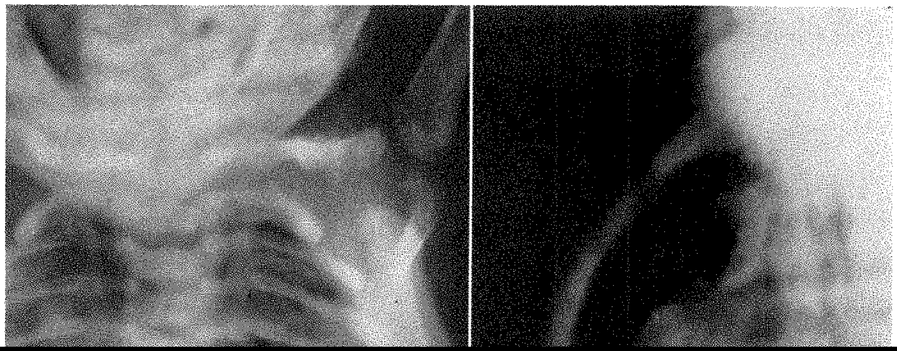

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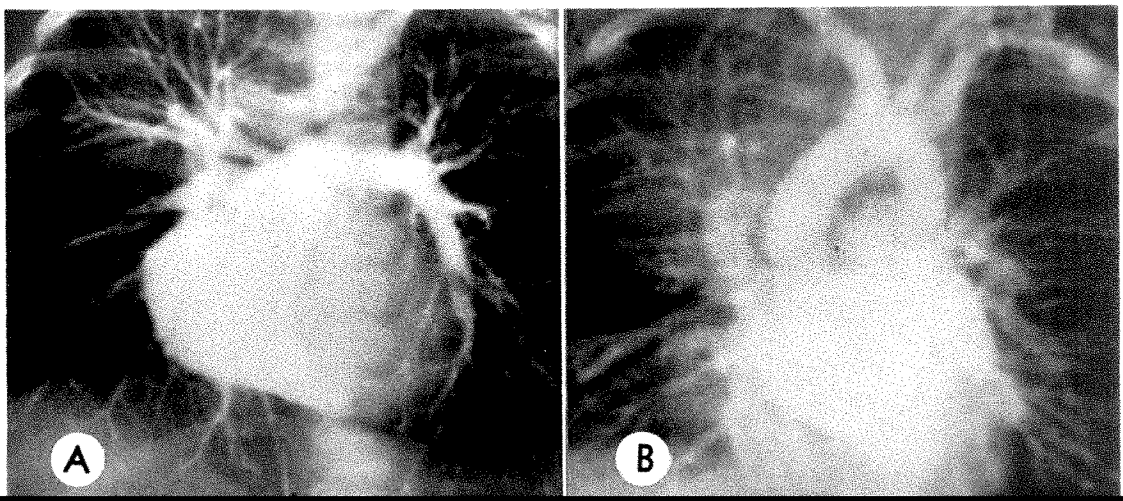

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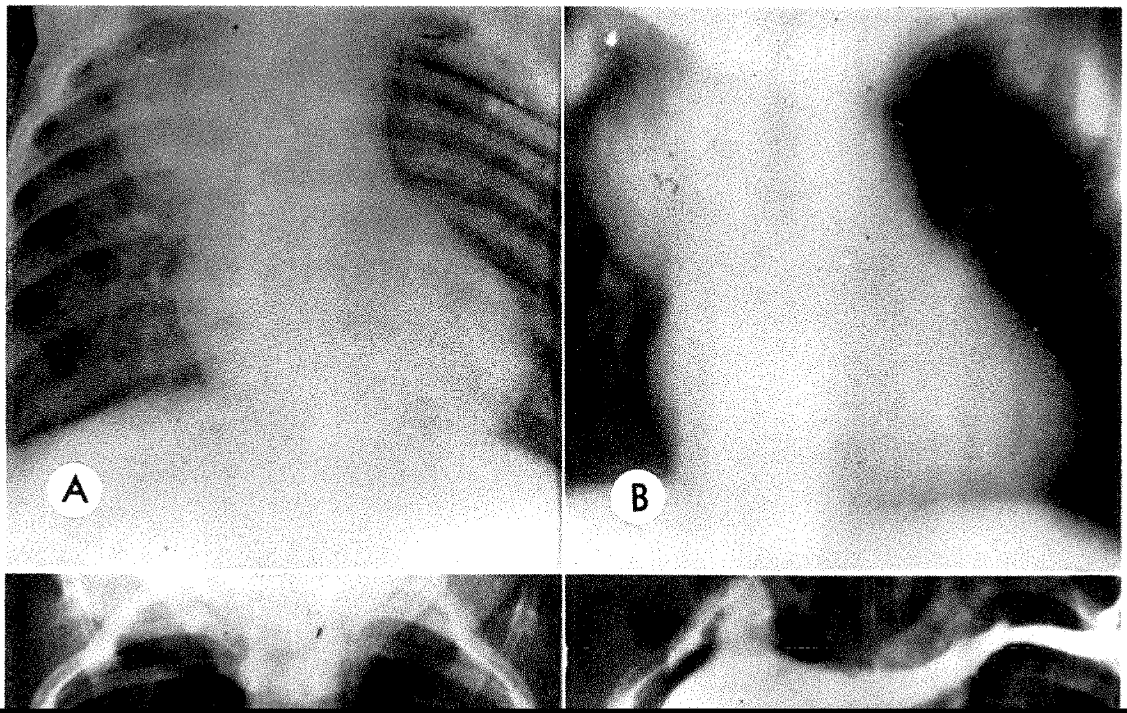

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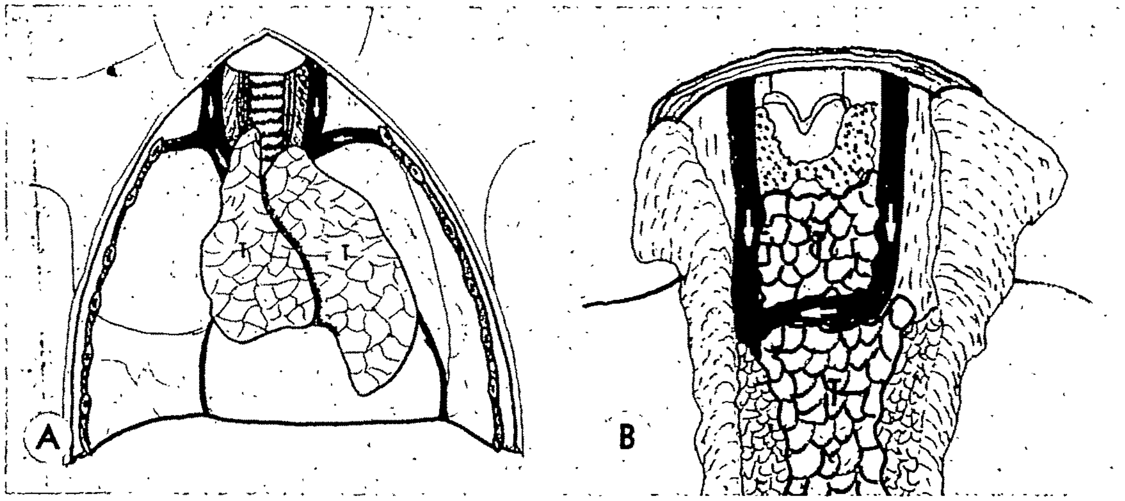

Fig. 15. Schematic representation of one of the most frequent anatomic types of hypertrophy of both lobes of the thymus. (*A*) Prevenous type. (*B*) Retrovenous type of the thymic gland. T = thymus.

and from the superior and inferior thyroid arteries. The veins end in the left innominate vein and in the thyroid veins.

Commonly, the gland is anterior to the great veins of the superior mediastinum (prevenous type), although Lucien and coworkers[10] described the "retro-venous type" (Fig. 15*B*). In this case the thymus is located behind the left innominate vein, and in part behind the upper segment of the superior vena cava. The occurrence is extremely rare; therefore, little has been published concerning it.

The normal position of the thymic gland before the great vessels is clearly demonstrated in Figure 16, which is a schematic section approximately at the level of the first intercostal space. This anatomic relationship explains why hypertrophy of the thymus does not displace the great vessels and the cardiac cavities, regardless of its size—a fact which is also illustrated in the levo-angiocardiogram presented by J. F. Hold[5] in a case of hypertrophy of the thymic gland.

In 1944[6] we described the "pseudocardiac hypertrophy of the thymus," in which both lobes of the gland cover the heart as a jacket. In some cases this type is associated with mild hypertrophy of the thymus, but in others the gland is very large mimicking a huge cardiomegaly. The cardio-thymic thoracic ratio can reach 0.71 or more.

In some patients the apex of the heart appears to be on the right side, simulating dextrocardia. In the vast majority of cases of this "pseudocardiomegaly" the right or left, or both, lobes can be identified because of the notched defect at the junction of the lower part of the thymic lobe with the heart.

During infancy a differential diagnosis between a huge hypertrophy of the thymus gland and pericardial effusion, in most cases, does not present great difficulty.[4]

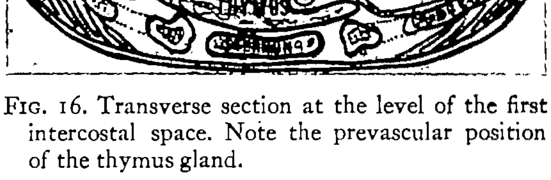

Fig. 16. Transverse section at the level of the first intercostal space. Note the prevascular position of the thymus gland.

Intrapericardial tumors and various types of mediastinal tumors are not easy to differentiate;[14–16,19–21,31] in these cases angio-

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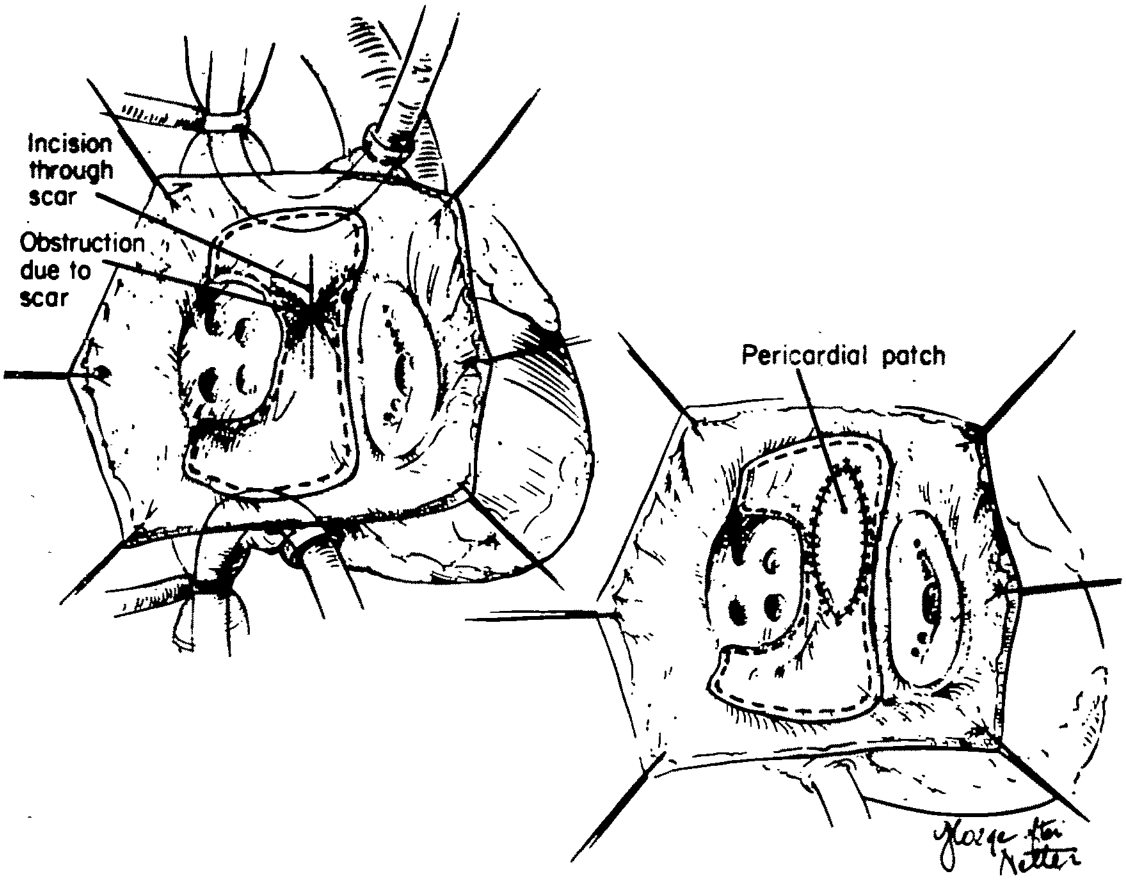

Fig. 4. Case 1. Operative findings and method of surgical repair are demonstrated. The baffle was found to be strictured in its upper portion just above the area overlying the mitral valve. A pericardial patch was sutured in place over the narrowed area.

modified Blalock-Hanlon procedure (creation of an A.S.D.) was performed with the incorporation of a pericardial patch into the posterior atrial wall over the atrial septal defect. The postoperative course was complicated by congestive heart failure for which the patient was digitalized. On the 5th postoperative day the thoracotomy wound dehisced and was subsequently treated with topical antibiotics and debridement. The patient gradually improved and was discharged at 25 days of age on maintenance digoxin. During the first year of life, the patient did quite well with only mild cyanosis on exertion and moderate ease of fatigability. Heart catheterization at the age of 1½ years demonstrated transposition of the great vessels with an effective atrial septal defect. The patient was recatheterized at the age of 4 years with essentially the same catheterization findings except for confirmation of the presence of a mild degree of subpulmonic stenosis.

At the age of 5 years he underwent complete correction using the Mustard technique. The postoperative course was complicated by bilateral pleural effusions. The effusion on the left required chest tube drainage of chyloserous fluid for 23 days. Paralysis of the left hemidiaphragm was noted. The patient was also treated for bacterial endocarditis because of a positive blood culture. Prolonged recurrent episodes of cardiac arrhythmia were noted with atrial-ventricular disassociation, seemingly aggravated by the digitalis. Through the hospital course enlargement of the liver was noted, which seemed to fluctuate from day to day as well as within the course of a day. This variable but persistent hepatomegaly uncontrolled by medical therapy led to the suspicion of an inferior vena cava obstruction. An inferior

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Mustard procedure is important since an operation to correct these hemodynamically critical lesions is mandatory in most cases.

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ANOMALIES OF THE ANTERIOR ABDOMINAL WALL: CLASSIFICATION AND ROENTGENOLOGY*

By EDMUND A. FRANKEN, Jr., M.D.
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NUMEROUS articles concerning umbilical anomalies and related malformations of the abdominal wall have been published since the treatise of Cullen in 1916,[5] but relatively few publications[9,11] concerning the pertinent roentgenology have appeared. In recent years a better understanding of the embryologic development of the abdominal wall has defined the relationship of the various defects to one another.[6]

The purpose of this paper is to correlate the embryology, pathologic anatomy, and applied roentgenology of the various abdominal wall anomalies encountered in clinical practice.

EMBRYOLOGY

The primitive embryo is disk shaped, with a dorsal ectoderm (continuous with the amnion), ventral endoderm (continuous with the yolk sac), and intervening mesoderm.[6] With further growth the mesoderm separates to form the extra-embryonic coelom (Fig. 1*A*). This cavity soon extends into the germinal disk, so that the ectoderm and endoderm with their associated portions of mesoderm are separated by the intra-embryonic coelom. The layer of mesoderm associated with the ectoderm is termed somatopleure; that with the endoderm, splanchnopleure.

Closure of the embryo body is accomplished by ventral folding of the lateral aspects of the embryo. Four folds can be distinguished: cephalic, caudal, and each lateral. The apex of the folds is the future umbilicus, and with their fusion the embryo body is closed except for attachments through the umbilicus (body stalk) (Fig. 1, *B* and *C*). Failure of formation of any of the folds results in the malformation of celosomia, *i.e.*, hernia of the anterior abdominal wall.[6]

An upper celosomia occurs with failure of formation of the cephalic fold. This condition results in omphalocele and defects of the anterior chest wall and diaphragm with ectopia cordis, as these structures arise from the cephalic somatopleure. The cephalic splanchnopleure, from which the heart and great vessels develop, is present, but accompanying cardiovascular malformations are common.

Middle celosomia occurs with failure of descent of the lateral folds, and the umbilicus remains wide and patent. This is the anomaly described as omphalocele.

With failure of formation of the caudal fold, a complex malformation known clinically as extrophy of the cloaca is seen. This consists of omphalocele, hindgut agenesis, and extrophy of the bladder with fistula of intestine to the extrophied bladder.

An anomaly which must be distinguished from omphalocele is that of gastroschisis. In this condition the somatopleure of the lateral fold adjacent to the umbilicus fails to differentiate. Secondary resorption of the overlying ectoderm results in a periumbilical defect through which bowel herniates. In contrast to omphalocele, no amniotic sac covers the herniated bowel.

Anomalies produced in later embryonic life include persistence of the vitelline duct connecting bowel lumen with the yolk sac (omphalomesenteric duct), and patent urachus (persistent allantoic canal between bladder and umbilicus).

Classification of the various congenital abnormalities of the umbilical region based on the above embryologic data is as follows:

I. Somatic Anomalies

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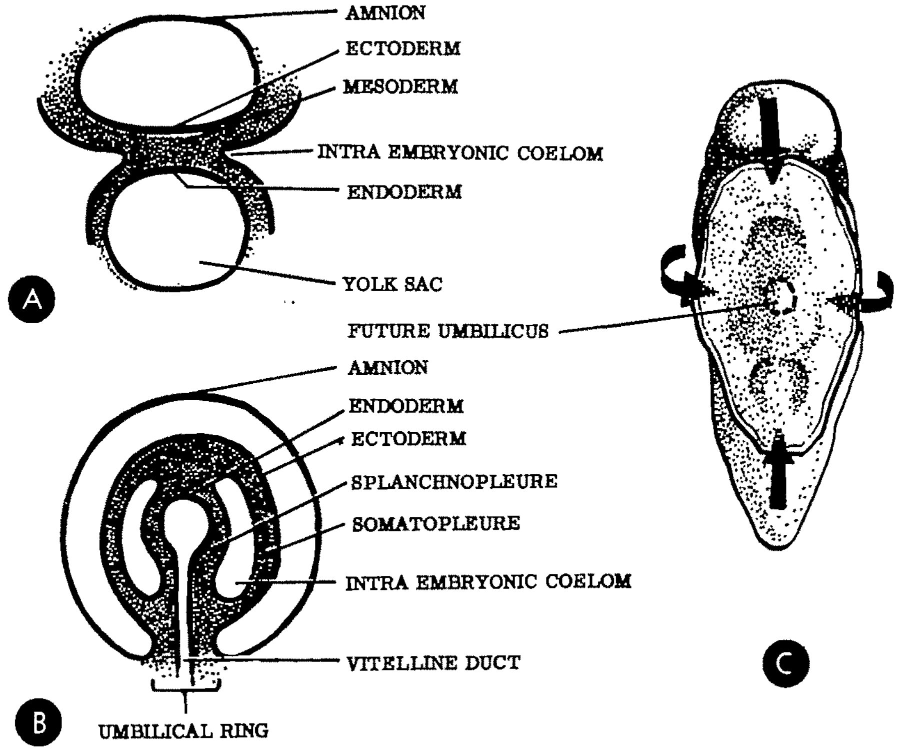

Fig. 1. (*A*) Diagrammatic cross section of the disk shaped embryo. The ectoderm is continuous with the amnion, the endoderm with the yolk sac. The intra-embryonic coelom divides the mesoderm into the somatopleure (adjacent to ectoderm) and splanchnopleure (adjacent to endoderm). (*B*) Ventral view of the embryo during closure of the body wall (after Duhamel). Four folds are seen: cephalic, caudal, and each lateral. These folds join at the future umbilical ring to produce the body stalk. (*C*) Cross section at the level of the umbilicus after ventral closure of the abdominal wall. The folds fuse at the umbilicus (body stalk). The ectoderm and somatopleure comprise the chest and abdominal walls and are separated from the endoderm and splanchnopleure by the coelomic cavity. The lumen of the future bowel communicates with the yolk sac through the body stalk by the vitelline duct.

  - A. Cephalic celosomia–midline defect of abdominal and chest wall, with deficient pericardium and diaphragm.
  - B. Lateral wall celosomia–omphalocele.
  - C. Caudal celosomia–extrophy of cloaca.
  - D. Lack of differentiation of lateral fold mesenchyme–gastroschisis.
- II. Umbilical Anomalies
  - A. Alimentary–complete or partial persistence of omphalomesenteric duct.
  - B. Urachal–complete or partial persistence of urachus.

DISCUSSION

CEPHALIC FOLD DEFECTS

A specific constellation of defects is seen in these patients.[3] Because of failure of medial migration of the sternum, there is an anterior defect in the chest wall, with a wide cleft in the lower sternum, and the heart is not covered by the rib cage. Dextroposition of the heart is invariably present, but true ectopia cordis does not occur in that the heart remains in the thorax. The clinical appearance of ectopia cordis results from lack of covering of the heart by rib cage and epigastrium. An anterior midline diaphragmatic defect occurs, with absence of pericardium in the same region. There is

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be seen in the lateral projection. The main portion of the omphalocele is located in the epigastrium. If malrotation with intestinal obstruction is present, the gas pattern should reflect this.

Treatment of this syndrome consists of omphalocele closure, repair of the diaphragmatic defect, and appropriate surgery for the heart disease present. The reader is referred to the excellent summary of Can-

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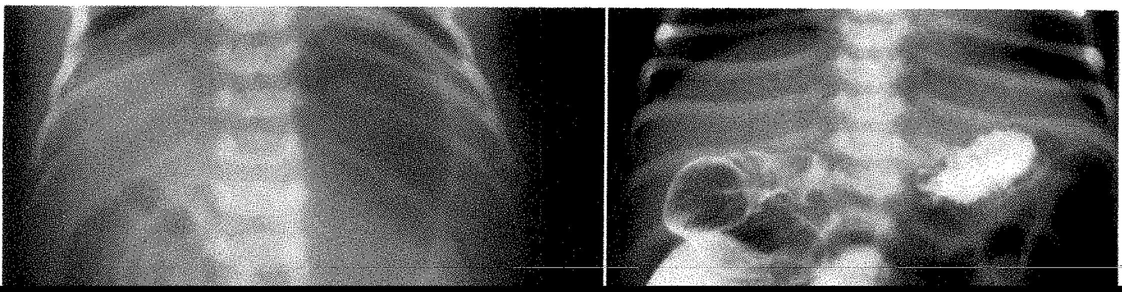
tion of liver in the peritoneal cavity. Less frequently other abdominal viscera including pancreas, spleen, and genital organs are found in large omphaloceles.

Attachment of the intestinal mesentery is abnormal in most cases of omphalocele, the base of the mesentery being fixed only by the superior mesenteric artery. This predisposes to midgut volvulus, and the duodenal bands accompanying malrotation are



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Gastrointestinal examination with bar-

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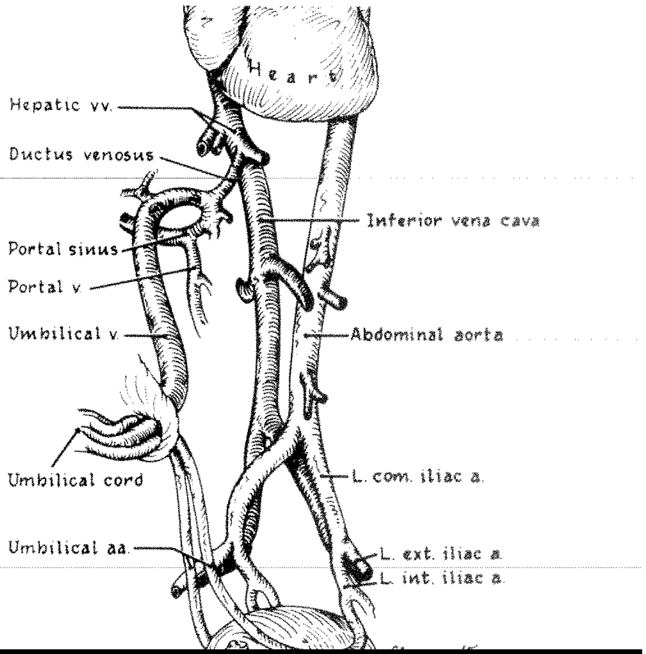
catheters were blindly introduced and then localized on roentgenograms exposed with a portable unit in the intensive care newborn nursery. Small, soft, plastic premature infant feeding tubes were used as catheters. All catheterization procedures were performed with strict sterile precautions.

Seventy-three neonates had only the umbilical vein catheterized, 12 had only the umbilical artery catheterized, and 25 had both the vein and an artery catheterized.

To prevent complications related to improper catheter placement, the tip of the umbilical vein catheter should lie in the inferior vena cava intrathoracically. The branchless ductus venosus is an acceptable but less optimal site. Baker *et al.*[1] advocate the right atrium as an alternate acceptable site. The tip of the umbilical artery catheter should lie in the distal abdominal aorta



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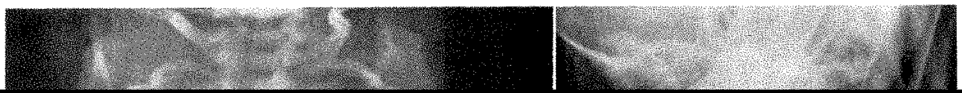

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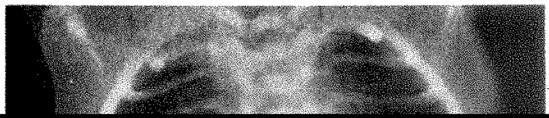
takes a gentle convex curve to the right of the mid sagittal line between T10 and L1 just before entering the ductus venosus



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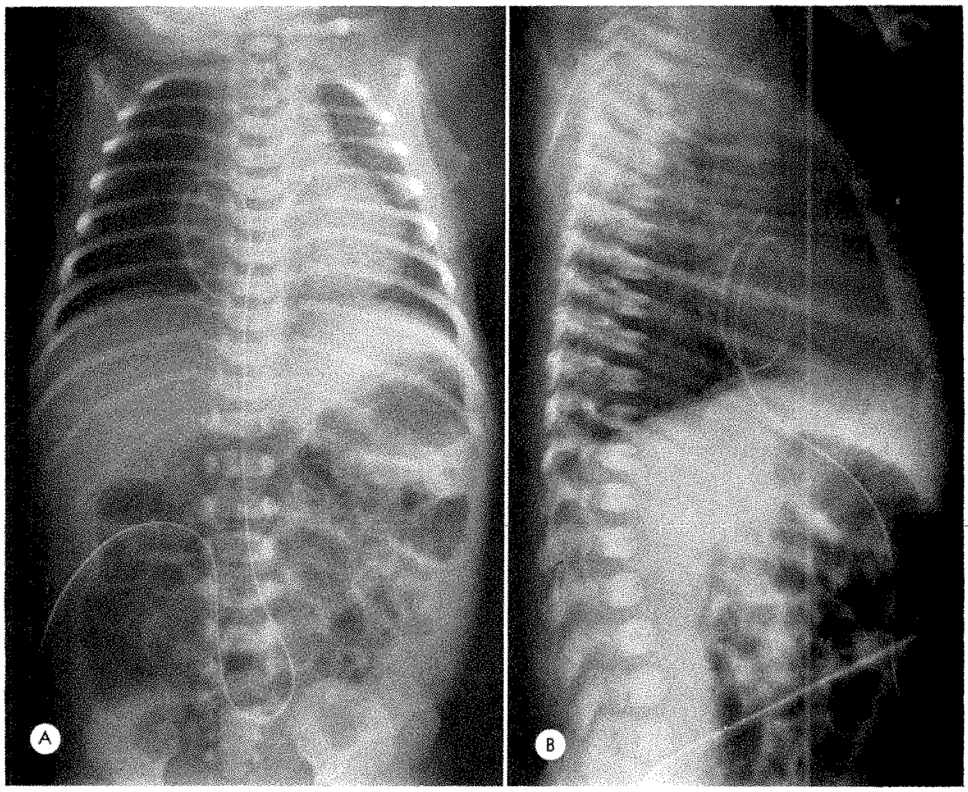

FIG. 5. Umbilical vein catheter. (*A*) In the anteroposterior projection the catheter extends from a coiled position in the right atrium, through the foramen ovale and left atrium, into a pulmonary vein above the left main bronchus. (*B*) In the lateral projection, exact localization of the catheter tip cannot be determined.

a "horseshoe" or "U-shaped" configura-

to be related to neonatal size, catheter size,

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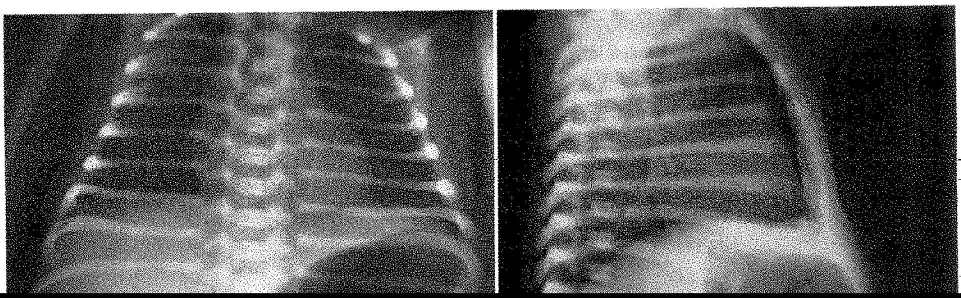

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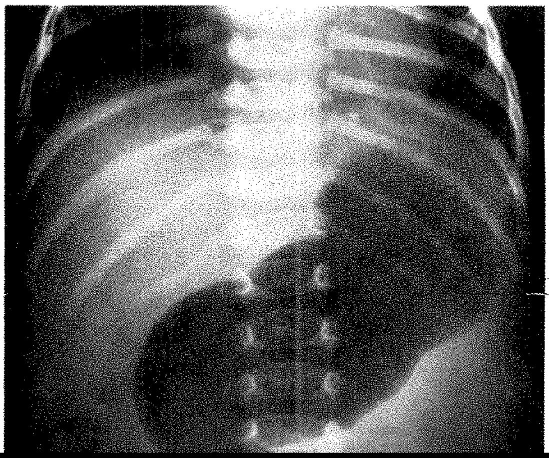
Table I

COMPARISON OF INCIDENCE OF PLAIN FILM ROENTGEN SIGNS BETWEEN PATIENTS WITH AND WITHOUT PYLORIC STENOSIS

	Non-pyloric Stenosis (100 Cases)	Pyloric Stenosis (100 Cases)
Stomach		
Inferior outline below L2	9	77
Maximum dimension greater than 7 cm.	7	57
Distention of 4 +	1	39

Thick Wall of Distal Gastric Antrum (Fig. 4). In the inferior portion of the pre-



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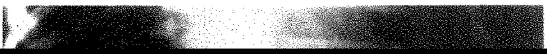
pyloric region, the thickened gastric wall is



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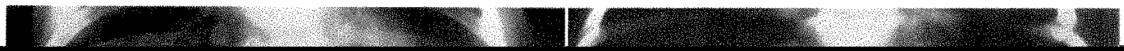

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without fluoroscopy in approximately half of the cases. It should be emphasized that pyloric stenosis can never be excluded on the basis of the plain film roentgenogram.

CONCLUSIONS AND SUMMARY

The plain supine abdominal roentgenogram was analyzed in 100 cases of proven pyloric stenosis, and the incidence of significant roentgen findings was compared with a control group of 100 clinically suspected cases in which pyloric stenosis was excluded.

Roentgen signs improving the radiologist's ability to predict pyloric stenosis were determined to center around a large hyperperistaltic stomach ("caterpillar" sign) descending into a relatively gasless lower abdomen. A gastric air outline measuring greater than 7 cm. and an outline extending below L2 were 2 of our most helpful criteria.

In clinically suspected pyloric stenosis, analysis of the plain film abdominal roentgenogram is helpful in individualizing the extent of further roentgenologic study. Approximately one-fourth of pyloric stenosis cases have enough corroborative evidence on their plain film roentgenograms to eliminate the need for further radiologic procedures which have at times led to needless radiation exposure. In the remaining cases the findings are helpful in deciding whether to use only overhead roentgenograms after barium administration or to supplement this with fluoroscopy.

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Vol. 112, No. 1

THICK GASTRIC FOLDS IN CHILDHOOD*

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RECENTLY the subject of Menetrier's disease in childhood has been reviewed with a total of 6 reported cases.[1] A single case report has also appeared.[12] The experience of Children's Hospital Medical Center (C.H.M.C.) is 3 cases of gastric rugal enlargement presenting with the symptomatology of Menetrier's disease. Two of these children had the benign course alluded to by others and 1 died of renal disease. An interesting finding pertaining to the possible etiology is presented.

REPORT OF CASES

Case I. J. De C., an 8 year old female, was admitted to C.H.M.C. on June 14, 1955, having had episodes of nausea and vomiting of increasing frequency for 4 months. Prior to admission the patient vomited small amounts of blood on several occasions. At no time was there any abdominal pain.

The past history revealed bouts of vomiting associated with poorly documented urinary infections since the age of 2 years. There was no history of allergy.

Apart from a slightly elevated blood pressure (B.P.) of 128/86 mm. Hg, the physical examination revealed no abnormalities. At the time of admission the laboratory data showed a hemoglobin of 10.7 gm. per cent. The total white blood cell count was 7,450 with no evidence of eosinophilia. Proteinuria of the order of 2+–4+ was present and the urine also contained several white blood cells as well as hyaline and granular casts. The nonprotein nitrogen (N.P.N.) was 20.9 mg. per cent (within normal limits). Serum protein and its fractions were not estimated at this time.

Excretion urography demonstrated no evidence of acute or chronic renal disease. The day of admission an upper gastrointestinal examination (U.G.I.) showed the esophagus to be normal, but there was marked irregularity of the whole stomach, especially on the greater curvature, due to large filling defects. The wall of the greater curvature was thickened as delineated by adjacent organs (Fig. 1; and 2). The duodenum and small bowel showed no abnormalities (Fig. 3).

The presumptive diagnosis of a diffuse gastric malignancy was made and the patient underwent a laparotomy 2 weeks later. The lesion was seen to extend from the antrum to within 6 cm. of the cardio-esophageal junction. The lesser curvature did not seem to be involved, but large lymph nodes were apparent in this area. Two were sent for frozen section and both were reported to be suspicious for lymphoma. Therefore, a 95 per cent gastric resection of Billroth I type was performed along with a splenectomy.

The pathologic findings consistent with Menetrier's disease are documented below. At the time of surgery the N.P.N. had risen to 37.9 mg. per cent with serum proteins of 4.22 gm. per cent and an albumin fraction of 1.84 gm. per cent. Following the operation the child fed poorly and lost weight. During the course of the next 3 to 4 weeks, ascites and left hydrothorax gradually developed. The investigations at this time showed unchanged serum proteins and albumin, but further elevation of the N.P.N. to 49.3 mg. per cent; and a 4+ albuminuria. The blood pressure was unaltered from the admission recording.

Some improvement in her condition occurred initially but she gradually became more hypertensive and showed evidence of increasing renal failure. She died at another hospital in uremia 6 months after her original admission.

Case II. M. D., a 9 year old girl, was admitted to C.H.M.C. in June of 1961 with "swelling of her abdomen and legs, as well as chronic abdominal pain."

She had been entirely well until May of that year when a bout of vomiting, abdominal pain,

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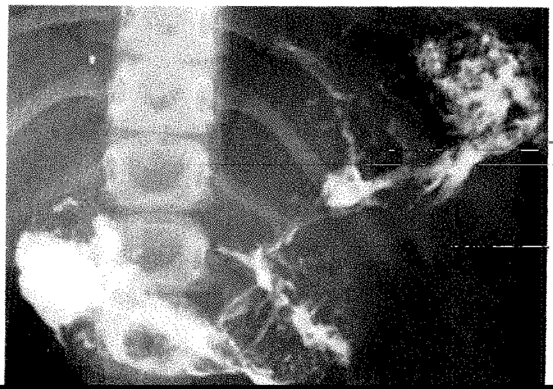


was admitted to C.H.M.C. for evaluation.

The past history was unremarkable. No history of previous allergy was elicited.

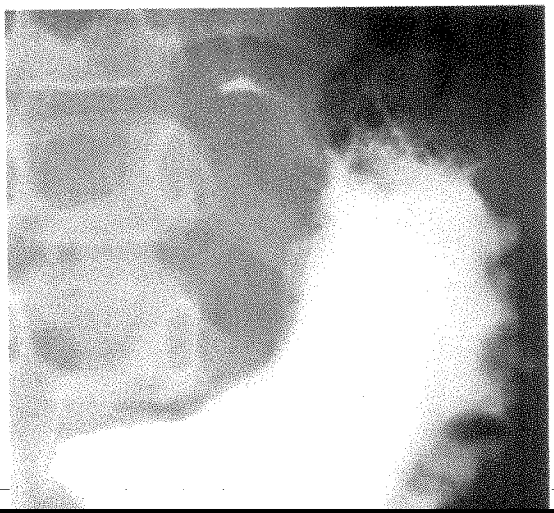
At physical examination the patient exhibited both anasarca and ascites. Her total serum protein (T.S.P.) was 3 gm. per cent with 2.1 albumin to globulin (A/G) ratio. Urine protein loss varied from 0–20 mg. per cent. At admission the girl had a 2 per cent eosinophil count on peripheral smear. The value rose to a high of 19 per cent 1 week after admission only to decrease gradually to 5 per cent 1 week later. Serum cholesterol levels were normal. Fasting

[redacted]

A follow-up examination 6 weeks after admission revealed a T.S.P. of 5.7 gm. per cent with an albumin total of 3.9 gm. per cent. She was readmitted 6 months later for rebiopsy at which time the only unusual finding was an elevated eosinophil count of 5 per cent. Another admission 1 year later for rebiopsy revealed no abnormal laboratory findings including a normal white blood cell count and differential. The 3 biopsies consistent with gastric mucosal edema will be discussed below.

The U.G.I. examination at that time, 18 months after the original C.H.M.C. admission, was entirely normal (Fig. 6).

Case III. P.C., a 7½ year old male, was admitted in April of 1969 with generalized edema,



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which time large edematous folds were seen on the greater curvature aspect of the stomach as well as some "inflammation of the mucosa" with several sites of bleeding.

The same day a laparotomy was performed. Externally, the jejunum appeared slightly thickened. Thick rugae were noted once again on the greater curvature of the stomach, sparing the antrum. Both jejunum and stomach were biopsied. The histology consistent with Menetrier's disease of these sites will be described.

The patient's hospital course was uneventful. Prior to surgery the serum albumin had risen spontaneously to 4.2 gm. per cent. However, postoperatively he did receive some intravenous albumin to raise the serum oncotic pressure and to promote wound healing.

After discharge the boy was seen again 1 month later in the clinic with a T.S.P. of 6.99 gm. per cent (albumin 4.28 gm. per cent). A repeat U.G.I. examination 5 months after admission showed an entirely normal stomach and small bowel (Fig. 10).

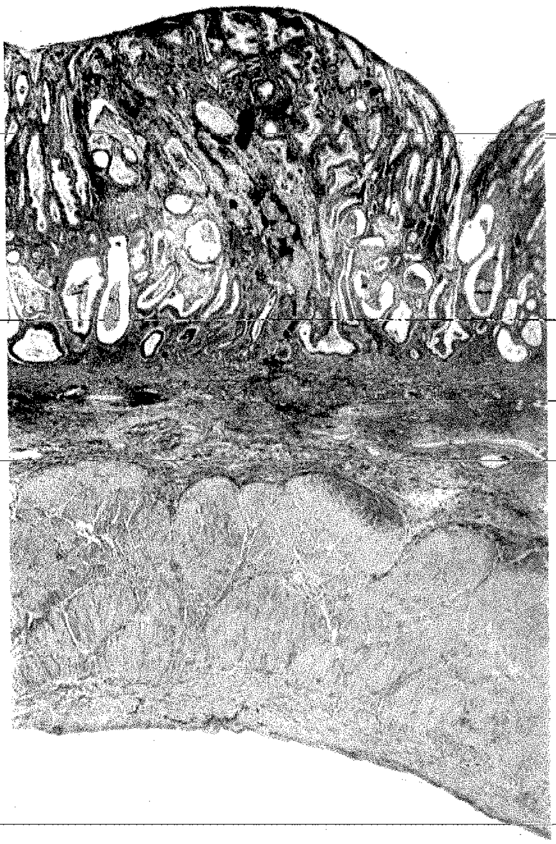

Fig. 11. Case I. Full thickness of gastric wall Sessile polypoid mucosal hyperplasia with microcystic retention changes are seen. (Toluidine blue-Eosin stain; 20×.)

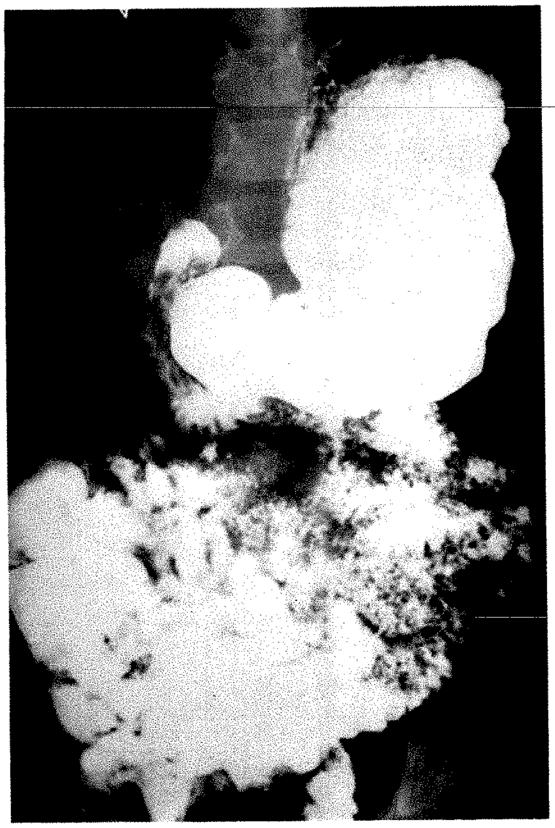

Fig. 10. Case III. Five months later the stomach has reverted to normal.

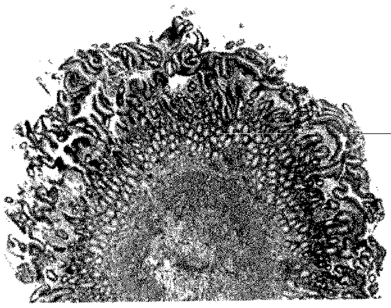

Fig. 12. Case II. First peroral aspiration biopsy of gastric mucosa. At this power some edema is apparent. (Hematoxylin and Eosin stain; 20×.)

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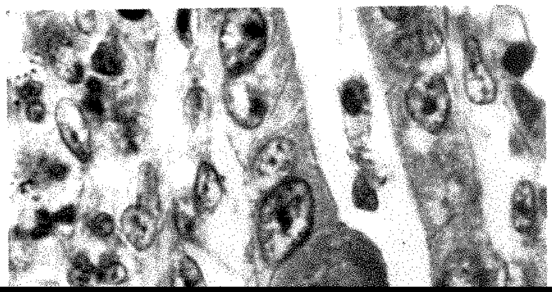
ening of the gastric wall, excessive secretions and a protein losing state. The original pathologic description of "polyadénomes en nappe" given by Menetrier in 1888[9] corresponds well with the histology in Cases I and III. Case II, while presenting a similar clinical and roentgenologic picture, was seen to exhibit only gastric mucosal



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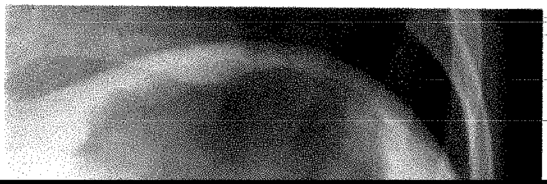

in childhood frequently progresses to a leukemic phase. Involvement of the stomach in this age group, however, is quite rare in both conditions.[3] In spite of the submucosal origin of lymphosarcoma it may

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were found in the gastric wall. The third patient had histology consistent with angioneurotic edema.

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VOL. 112, No. 1

CONGENITAL PREDUODENAL PORTAL VEIN*

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CONGENITAL preduodenal portal vein is a rare anomaly with significant surgical implications for the pediatric and adult population.

This report documents the clinical and anatomic findings in 2 children in whom a preduodenal portal vein was found. In the first case the anomalous vein was found during surgical exploration for diagnosis and correction of biliary atresia. The experience gained from the first case permitted a suggested roentgenographic preoperative diagnosis in the second child. Understanding the embryologic development of the anomaly, as well as proper roentgenologic documentation, may permit a preoperative diagnosis.

REPORT OF CASES

CASE I. (L. W.)

First Admission.

This white female patient was first admitted to Barney Children's Medical Center at the age of 2 months, with a history of jaundice since birth. There was also a history of dark urine and light colored stools. The physical examination revealed an enlarged liver and a palpable spleen. The laboratory studies at this admission included a white blood cell count of 9,600 per mm.^{3}; hemoglobin 12.2 gm. per 100 ml.; hematocrit 34 per cent; reticulocytes 6.8 per cent; normal platelets and normal red cell morphology. The liver function studies were as follows: SGOT 166 sigma units; SGPT 80 sigma units; LDH 940 Wroblewski units; prothrombin time 72 per cent; total bilirubin 5 mg. per 100 ml., unconjugated fraction 4 mg. per 100 ml.; thymol turbidity 1.8 units; alkaline phosphatase 20.2 units; direct Coomb's test negative. The urinary findings included a urinary urobilinogen of .25 units; the urinary sediment revealed 7 white blood cells per high power field, and an occasional red cell.

Roentgenologic Findings. October 7, 1967 (chronologic age 3 months). Anteroposterior roentgenogram of the chest showed mild passive congestion in the central lung fields bilaterally. Roentgenograms of the hands and wrists demonstrated no rachitiform findings. The skeletal maturity was less than 3 months. Gastrointestinal examination revealed gastric distention with extrinsic impression on the stomach by hepatosplenomegaly. Proximal duodenal angulations superiorly and medially were present (Fig. 1). The second portion of the duodenum also had a loop anomaly resulting in duodenal inversion (Fig. 2). Initial duodenal distention was incomplete with 60 minute proximal gastric retention (Fig. 3). Better filling of the duodenum demonstrated malrotation with right subhepatic duodenal and jejunal position. The jejunal location was lower in the right upper quadrant than the typical malrotation. Later timed roentgenograms revealed the right-sided small intestinal loops to have a malabsorption pattern, secondary to cirrhosis.

Operation. Exploratory laparotomy and liver biopsy were carried out. The liver was moderately enlarged with a nodular surface interpreted as biliary cirrhosis. There was no gallbladder and no extrahepatic biliary ductal system. An anterior placement of the portal vein across the second division of the duodenum was found, and the portal vein traced to the hilus of the liver. There was no evidence of duodenal obstruction and no corrective surgery was performed. The pathologic diagnosis of the liver biopsy was biliary cirrhosis secondary to congenital biliary atresia. The patient was discharged in an unchanged condition.

Second Admission.

The patient was re-admitted 4 months later, because of increasing respiratory distress. There was increasing shortness of breath and grunting respirations. Abdominal size had increased and the jaundice continued in varying degrees. Physical examination at this time revealed an emaciated, jaundiced, 7 month old infant with respiratory distress. The abdomen was tense with fluid, and there was an increase in venous pattern over the anterior abdominal wall. Laboratory data revealed a white blood cell count

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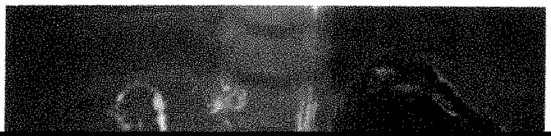

manifestations in the central lung fields bilaterally, and an increase in pericardio-cardiac size had occurred. Rachitiform bone changes had developed in the ribs. The abdomen showed

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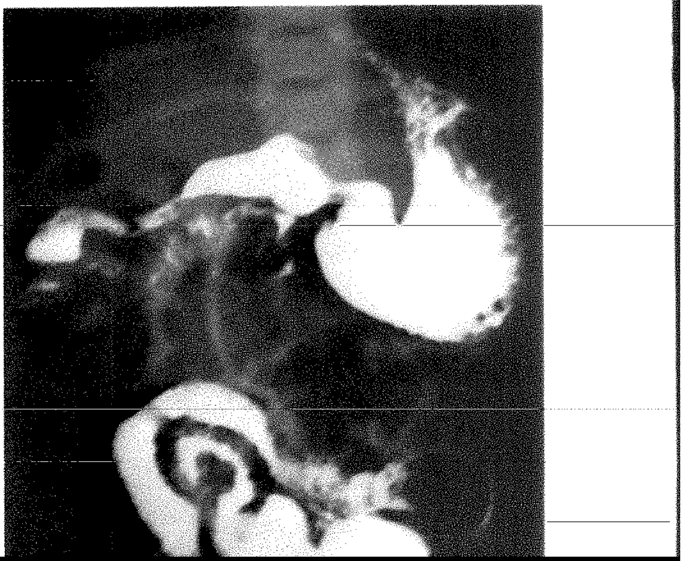
Roentgenologic Findings. May 8, 1968 (chronologic age 10 months). Roentgenogram of the chest showed that the manifestations of congestion were complicated by infiltration. A further increase in pericardiac size had occurred. The upper abdomen became additionally distended with ascitic fluid.

Course. The patient expired at the age of 22 months.

Autopsy. The anatomic diagnosis included: (1) congenital atresia of the hepatic biliary ducts with biliary cirrhosis and obstructive jaundice (Fig. 6); (2) malrotation of the small intestine and colon with anomalous location of the portal vein anterior to the duodenum and partial annular pancreas (90 per cent of the circumference) (Fig. 7; and 8); and (3) multiple spleens.



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incomplete ring constricting the proximal third of the second portion of the duodenum. A common mesentery was present with the jejunal loops central or right central in location.

DISCUSSION

HISTORIC REVIEW

Nineteen cases of this anomaly have been reported. In 1963, Renner and Child[9] reported a case of preduodenal portal vein, in association with biliary atresia and also

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signs of biliary rickets, malnutrition, and edema will also be present.

Anomalous location and malfixation of the right colon and cecum will contribute to

With recognition of dextrocardia as part of a multiple congenital anomaly complex, including situs inversus, an increasing awareness by the pediatrician and later by

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that can be associated with preduodenal portal vein could have been double portal vein and congenital stenosis of the portal vein, which would have also added to the surgical hazard. Case II likewise showed no obstruction due to the preduodenal portal vein. The intrinsic membrane of the duodenum was the cause of incomplete obstruction. The extrinsic bands were the cause of the duodenal angulation.

SUMMARY

Preduodenal portal vein may occur as an isolated event and result in no symptoms, or be found at autopsy as an incidental finding. It is much more likely to be part of an anomaly complex.

The clinical-roentgenologic correlation of the following features should suggest preduodenal portal vein:

1. Visceral malrotation
    a. dextrocardia

A roentgenographic diagnosis is emphasized in this report to reduce surgical hazards which may occur during procedures for relief of duodenal obstruction, or as may occur during attempted liver transplants.

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SEGMENTAL DILATATION OF THE COLON*

By ALFRED A. de LORIMIER, M.D., STEPHEN R. BENZIAN, M.D.,
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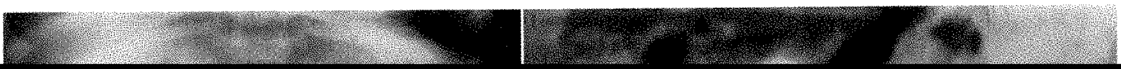
SEGMENTAL dilatation of the colon, [redacted]

and a few hard pieces of stool were in the am- [redacted]



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from the cecum to the descending colon. As

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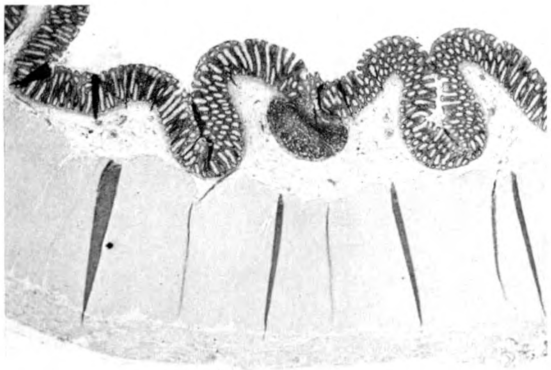


FIG. 4. Full-thickness section of the transverse colon. There is greatly hypertrophied circular and longitudinal muscle.

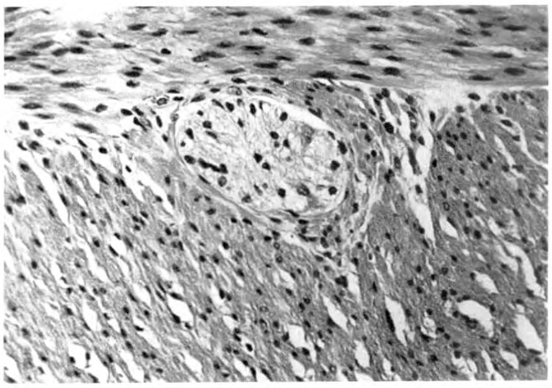
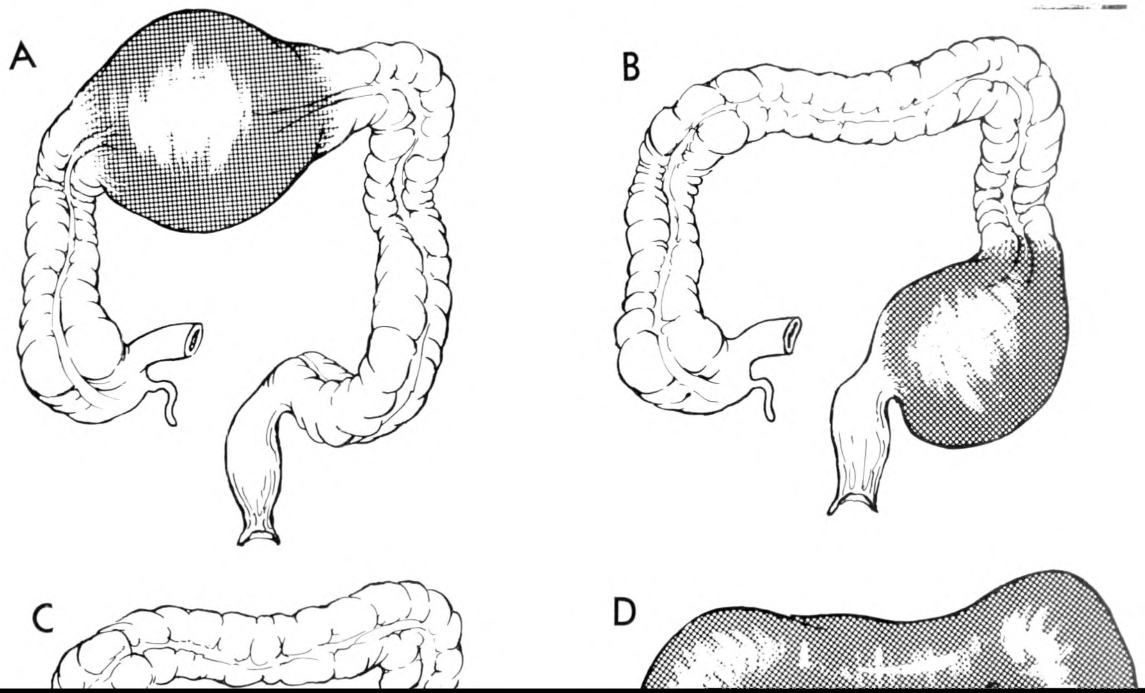


FIG. 5. Histologic section of nondilated sigmoid colon. Normal ganglial cells and neurofibrils are present.



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mal ganglion cells were present in the rectum and these children responded well to colostomy. Subsequently, a Swenson abdominoperineal pull-through procedure was performed and good results were obtained. Hypertrophy of the muscularis in the dilated colon was not described in these cases. Colostomy, however, may have protected the bowel from hypertrophy.

The roentgenographic distinction between segmental dilatation of the colon and Hirschsprung's disease may be difficult. Barium enema examination in Hirschsprung's disease should reveal a normal or small caliber of the rectum. The aganglionic bowel does not distend readily, and irregular, serrated contractions should be identified.[4] Conversely, in our patient with segmental dilatation of the colon, the distal colon and rectum were distensible and the irregular contractions were not seen. In both entities the bowel wall of the proximal colon with normal ganglion cells may be thickened as a result of muscular hypertrophy. In both diseases, evacuation of barium from the colon may be considerably delayed. The distinction between segmental dilatation of the colon and Hirschsprung's disease should be resolved by biopsy of the rectal muscle. Histologic examination should establish the presence or absence of ganglion cells and normal or abnormal neurofibrils.

The cause of segmental dilatation of the colon is unknown. Hypertrophy of the muscularis and dilatation of the bowel might be expected if mechanical obstruction were present distally. However, no obstructive lesion has been observed in these patients. The distal bowel has been distensible and peristaltic function after resection of the dilated segments has been normal. Since each patient responded well to resection of the dilated bowel, this abnormal segment would appear to have been the cause of constipation. In our patient, the circular muscle layer of the dilated bowel appeared more hypertrophied than the longitudinal muscle layer. The number of muscle cells was increased rather than individually enlarged. Although normal-appearing ganglion cells were present throughout the colon in these patients, new histochemical techniques for identifying adrenergic and cholinergic nerves in the dilated segment may reveal a neurogenic cause for this disease. Since the symptoms of constipation and abdominal distention developed in early infancy, segmental dilatation may be a congenital anomaly.

SUMMARY

Segmental dilatation of the colon is a rarely described disease in which a variable length and portion of the colon is greatly dilated. The muscular wall of this segment is hypertrophic and ganglion cells are present throughout the colon and rectum. These patients have constipation which may resemble that of Hirschsprung's disease. The dilated segment, however, rather than the rectosigmoid colon should be resected.

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HIRSCHSPRUNG'S DISEASE*

PROBLEMS IN THE ROENTGEN INTERPRETATION

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THE classic roentgenographic features of Hirschsprung's disease have been well documented.[5] They include narrowing and spasm of the aganglionic distal segment of bowel in continuity with more dilated proximal, normally innervated bowel. Classic findings are not always present, especially in the neonate.[8,13] Some roentgenographic features in the difficult cases may exist, however, which either reflect underlying Hirschsprung's disease, or mask or mimic it.

Seven cases are reported, all of which were considered to possibly have Hirschsprung's disease. The details of the histories, roentgenographic findings and confirmed diagnoses are discussed.

It is the purpose of this paper to present case material which demonstrates some problems leading to difficulty in making or excluding the diagnosis of Hirschsprung's disease.

REPORT OF CASES

CASE I. R.C. This white male was born on November 27, 1968, following an uncomplicated pregnancy and delivery. After the first 36 hours only scanty quantities of meconium had been passed and his abdomen became distended. The abdominal plain roentgenograms suggested a low bowel obstruction. Barium enema roentgenograms demonstrated no significant rectal spasm but much meconium packed into the rectum (Fig. 1, *A* and *B*). The radiologist had had little experience with this problem and obtained spot roentgenograms only in the anteroposterior position. His diagnosis was "meconium plug syndrome." Some meconium with a yellow-green "phrygian cap" or leading plug passed which did support his contention. Consultation with other radiologists alerted him to a possible localized area of anorectal spasm and hence the possibility of Hirschsprung's disease. A repeat study 10 days later demonstrated relatively persistent anorectal spasm (Fig. 1, *C* and *D*). After the second examination a correct diagnosis of Hirschsprung's disease was made and confirmed by biopsy.

Initial impression. Meconium plug syndrome. *Final diagnosis.* Hirschsprung's disease.

CASE II. B.L. This white male infant was born at Michael Reese Hospital on November 14, 1968. He passed no meconium in the first 24 hours of life and was becoming distended. A barium enema examination demonstrated no spasm, no differential bowel caliber, and meconium in the rectum all the way to the anorectal junction (Fig. 2, *A* and *B*). After instillation of barium and roentgenography, evacuation was allowed to proceed. There was no explosive-type evacuation and one or two large meconium boluses were passed. The distention subsided and the infant was returned to the nursery with the diagnosis of meconium plug syndrome. He experienced no reported difficulty under expert care while in the nursery. He was discharged 5 days later. The child was readmitted on December 11, 1968. He had done well at home for 2 weeks having 4 to 5 pasty yellow stools per day, spontaneously. At 2 weeks of age he did have a 40 hour period without bowel evacuation. This was relieved by oral diluted prune juice. Three days prior to his admission at 1 month of age he had gone 72 hours without bowel evacuation. A barium enema examination now showed the classic findings of Hirschsprung's disease (Fig. 2, *C* and *D*).

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Case v. E.H. This $7\frac{1}{2}$ month old white female entered Children's Memorial Hospital with a diagnosis of possible megacolon made at the referring hospital. Barium enema examination demonstrated findings consistent with Hirschsprung's disease (Fig. 5, *A* and *B*). At surgery, while under anesthesia, the patient was re-examined. Digital examination of the rectum disclosed a rectal stricture 4 cm. from the anal orifice. This correct diagnosis was made prior to the definitive pull-through procedure of Swenson. After the procedure further investigation suggested that the child had some difficulty in the newborn period but not of any significant nature. Since the resected bowel demonstrated some inflammatory changes it is

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consultation suggested some exposure to significant traumatic episodes and supported the diagnosis of psychogenic constipation. On the final admission, 2 years later, a repeat barium enema examination (Fig. 7, *A–C*) and biopsies were obtained. A rectal myomectomy and anal sphincterotomy were performed. The biopsies showed no ganglion cells within 1 cm. of the anorectal junction, a few ganglia at 2 cm. proximally, and scattered ganglia at 3 cm. from the anorectal junction.

Initial impression. Psychogenic megacolon.
Final diagnosis. Short segment aganglionosis.

DISCUSSION

The diagnosis of Hirschsprung's disease in the older child generally presents little difficulty. All the classic roentgenographic features can be defined with routine barium enema study. These findings include a narrow segment of distal colon (the aganglionic portion) and the wider, usually fecally impacted, normally innervated proximal bowel. Some difficulty in demarcating the true site of normally innervated bowel from bowel without myenteric plexuses may occur. This is because the impacted fecal matter may be forced into the more distal abnormal bowel, widening the caliber.[11]

In the neonate and infant and in a few older children with constipation the diagnosis may be easy to make; it may also be extremely confusing.

Careful attention must be given to the details of the barium enema examination. After an infant-size plastic rectal tip is

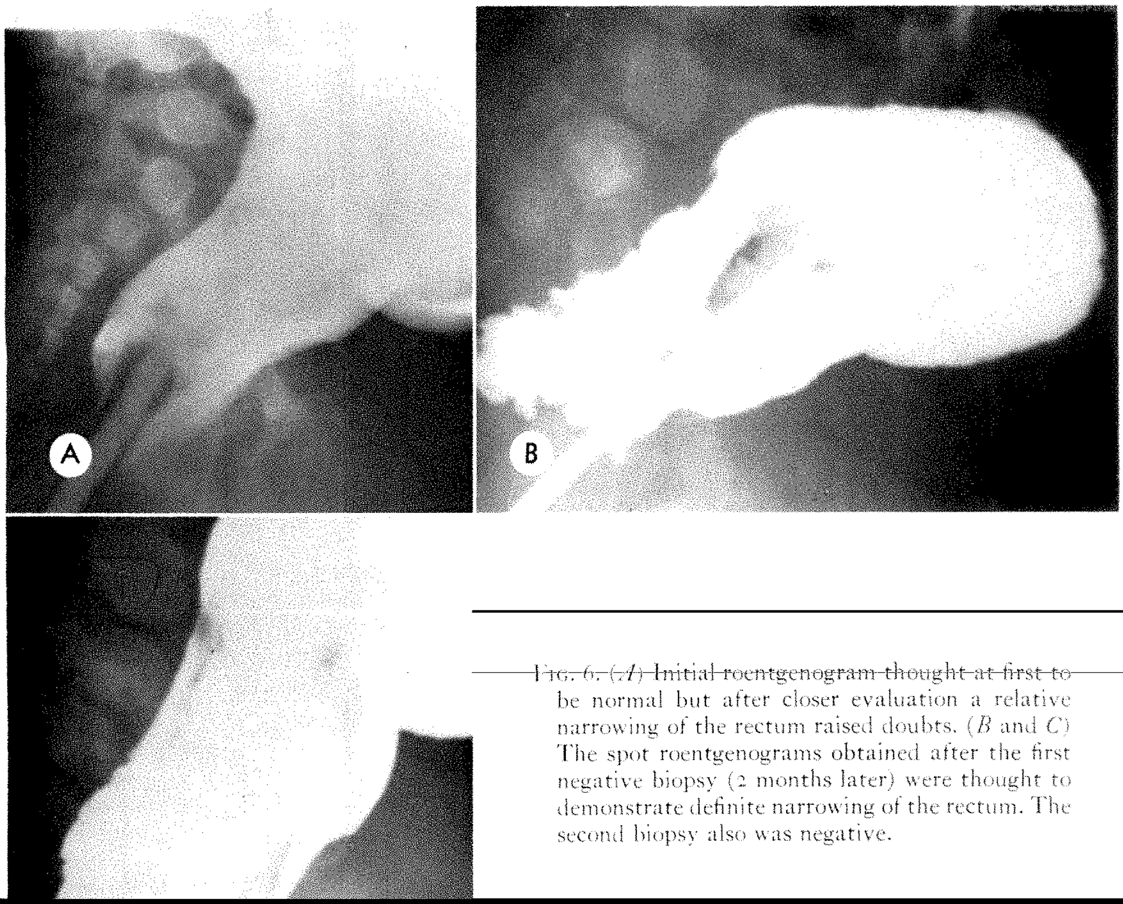

FIG. 6. (*A*) Initial roentgenogram thought at first to be normal but after closer evaluation a relative narrowing of the rectum raised doubts. (*B* and *C*) The spot roentgenograms obtained after the first negative biopsy (2 months later) were thought to demonstrate definite narrowing of the rectum. The second biopsy also was negative.

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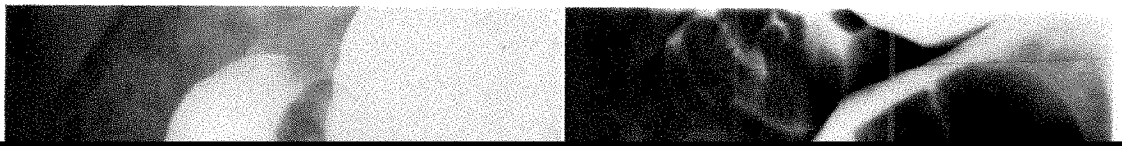

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Bardex catheter is never used as the roentgenographic findings may be obscured.

The barium is then instilled while the patient is in the frontal position. Only a small amount of barium is delivered initially. The patient is then rolled onto the left side or into the left posterior oblique position and observed again.[2] More barium may be needed but flooding rapidly is to be avoided. Spasm and/or narrowing of the aganglionic segment yields the classic picture in the neonate, especially when the more proximal portion of bowel is seen with its nonspastic, normal caliber juxtaposed. The spastic portion may show irregularity of the mucosa suggesting early submucosal inflammation. The authors believe that this picture of mucosal irregularity may be present in the absence of associated inflammation. It is wise to initiate the patient's care rapidly, however, as submucosal inflammation in an infant with Hirschsprung's disease may precede and cause sepsis and death.[3] The aganglionic segment may be of any length but it may be assumed that wherever the aganglionosis begins, all the bowel from that point distal is devoid of normal neurogenic elements. Evidence to the contrary has been reported (skip areas) but these cases are extremely rare.[4]

Difficulties in diagnosis occur when the classic findings are not present. In Case IV the classic fluoroscopic image presented but only for an abbreviated period of time. Spasm of the rectosigmoid was definitely present. It was not demonstrated in the descending colon. Although the findings were only temporarily observed (about 5–6 seconds) it was believed that the appearance was that of Hirschsprung's disease. The remainder of the colon demonstrated a malrotation but intrinsically the colon was otherwise normal. An upper gastrointestinal examination at this time would have demonstrated a volvulus, and possibly devitalization of bowel might have been considered. The patient was operated upon immediately, however, and much inflammatory fluid was found in the peritoneal cavity. An early peritonitis was also found as well as the findings noted above. It is believed that irritating inflammatory fluid and the peritonitis caused spasticity of the rectosigmoid noted at fluoroscopy, thus creating the findings interpreted incorrectly as Hirschsprung's disease.

Masking of the findings occurred in Case I. Initially the examiner obtained only frontal spot roentgenograms and interpreted the findings as a meconium plug syndrome. The patient did evacuate the barium and much meconium, as stated in the case presentation. A repeat examination done within 10 days of the first examination clearly demonstrated the classic findings of Hirschsprung's disease. Not obtaining oblique and lateral roentgenograms and observing the patient in these positions is the pitfall to be guarded against.

Absence of the findings in Case II cannot be interpreted as a pitfall. The patient did have a classic history for either meconium plug syndrome or Hirschsprung's disease. Clearing of the symptoms following the barium enema examination, absence of a spastic segment and elimination of the symptoms for 1 month supported the discharge diagnosis of meconium plug syndrome. The infant also did not have explosive-type bowel evacuations of watery stool as is frequently seen in Hirschsprung's disease and/or colitis and did not become distended. The symptoms recurred only after 1 month and the correct diagnosis of Hirschsprung's was made over the telephone by the history (albeit unusual), by physical examination (absence of stool in the rectum), and visually by barium enema study. This case suggests that Hirschsprung's disease need not cause daily difficulties in the neonatal period and may occur in an infant who initially presented a classic meconium plug syndrome. That infants with meconium plug syndrome may later have Hirschsprung's disease has been cited by other authors.[7]

Colitis with or without sepsis can cause spasm of the bowel. The neonate is seldom, if ever, the object of classic idiopathic ulcerative colitis. Generally the neonate with colitis has an underlying sepsis and/or error in the diagnosis of Hirschsprung's

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sprung's disease.

Anorectal narrowing and/or spasm should strongly suggest, but is not pathognomonic of, aganglionosis. It may be seen in patients with sepsis and secondary colitis, infectious peritoneal fluid and peritonitis, and in patients with structure formation.

The meconium plug syndrome may be the earliest manifestation of a patient with Hirschsprung's disease and may mask it entirely. Findings of Hirschsprung's disease may be difficult to recognize or be nonexistent in the first month of life, however, when the meconium plug syndrome is encountered.

The tapered anorectum, as noted on the barium enema examination, may mimic Hirschsprung's disease, whereas the patient with true "short segment" aganglionosis may demonstrate no aganglionic zone on barium enema study and be labeled a "psychogenic" problem.

Adequate biopsy, including rectal myomectomy, in patients with suspicious aganglionosis is necessary for complete evaluation. It may be therapeutic in both cases of short segment aganglionosis and "psychogenic megacolon."

SUMMARY

Pitfalls in the roentgenologic diagnosis of Hirschsprung's disease occur, especially in the neonate and infant.

Seven cases are reported, each demonstrating where errors in the diagnosis have been made. In 4 cases Hirschsprung's disease was suggested as the initial diagnosis, but was not. In 2 cases the lesion was initially called meconium plug syndrome, but was aganglionosis. The seventh case was roentgenographically considered to have psychogenic megacolon, but was found to have a short segment type Hirschsprung's disease by biopsy.

The unique details of each case are presented.

An analysis of the findings shows some of the causes for misinterpretation of the roentgenographic investigation. The misinterpretation led to incorrect diagnoses both in cases of true aganglionosis and those later proved not to have Hirschsprung's disease.

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ROENTGEN APPEARANCE OF THE EXCLUDED COLON AFTER COLOSTOMY FOR INFANTILE HIRSCHSPRUNG'S DISEASE*

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ROENTGEN examination is probably the most important diagnostic study for early detection of Hirschsprung's disease, and the roentgen signs of congenital aganglionic megacolon in infancy have been described in detail.[1,6,13] Early recognition of the disease and appropriate surgical treatment have sharply reduced morbidity and mortality.[9,18]

Colostomy above the aganglionic segment is the treatment of choice in neonatal Hirschsprung's disease; a definitive procedure (abdominoperineal pull-through) is then performed at a later date, usually at the age of about 1 year, depending on the general condition of the patient and the progress of improvement.[9,18,19]

There are 3 main types of abdominoperineal pull-through procedures: Swenson's, Duhamel's and Soave's. In all, the aganglionic segment is resected and the proximal colon is brought down and anastomosed to the anus.[14,18,19]

Postoperative complications are not uncommon, and as experience increases more infants are now referred for roentgenologic examination after surgery. James *et al.*[14] described postoperative shortening of the colon, straightening of the descending colon, inferior displacement of the splenic flexure, anterior rectal pouch with the Duhamel procedure, and straight and narrow distal colon with wide presacral space after Soave's operation. We have noted rigidity, poor distensibility and narrowing of the colon to occur after colostomy. In addition, striking mucosal changes have been observed, such as thickening and irregularity of folds, with distinct nodularity, often giving a "cobblestone" pattern to the mucosal relief. These changes involve the segment of the bowel distal to the colostomy and form the substance of the present report.

CLINICAL MATERIAL AND OBSERVATIONS

Between 1964 and 1969, 9 patients with Hirschsprung's disease have had serial roentgenographic examinations before and after surgery (Table I). In 4 patients, the

TABLE I

CLINICAL, ROENTGEN AND SURGICAL PATHOLOGIC FEATURES OF 9 PATIENTS OPERATED FOR HIRSCHSPRUNG'S DISEASE

Patient	Sex	Age at Diagnosis	Enterocolitis	Contrast Enema, After Operation		Gross Changes at Surgery		Histology		Definitive Surgery Type
				Narrow, Non-distensible Colon	Nodular Mucosa	Redundant Folds	Polyps	Lymphoid Hyperplasia	Active Inflammation	
1. J.L.	M	1 mo.	+	+	+	+	+	+	−	D
2. W.Y.	F	3 da.	+	+	+	+	+	+	−	D
3. J.D.	M	2 mo.	+	−	+	+	+	+	+	S
4. Y.L.	F	2 mo.	+	−*	+*	+	+	+	−	D
5. E.S.	M	1 wk.	−	+	−	−	−	−	−	D
6. G.G.	M	6 mo.	+	−	−	−	−	−	−	D
7. C.P.	M	2 mo.	+	+*	+*	+	+	+	−	So
8. H.F.	M	2 wk.	−	+	±	+	±	−	−	So
9. M.N.	F	6 mo.	−	+*	+*	−	+	+	−	So

* Changes first noted after simple colostomy. In all other cases, not marked by asterisk, contrast study was first performed immediately after definitive surgery.

D-Duhamel's, S-Swenson's, So-Soave's procedure.

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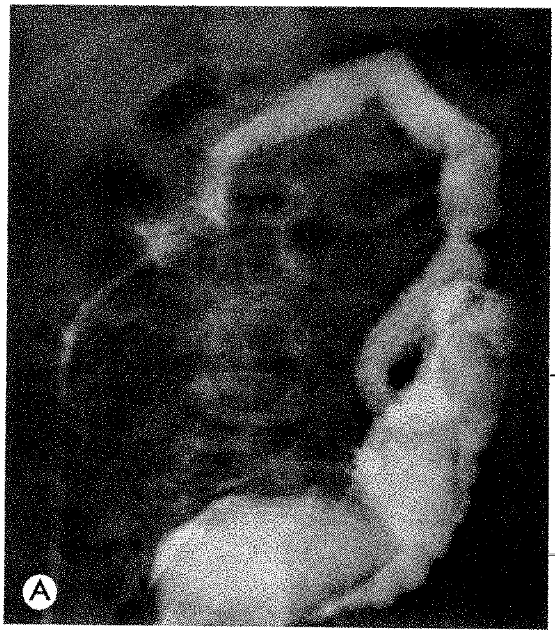

FIG. 4. Case 9. (*A*) About 3 months after colostomy the colon shows typical "cobblestone" pattern.

lymphoid polyposis.[4,5,16] Its etiology is unknown. Ability to recognize this condition and good contrast enema studies have shown that lymphoid hyperplasia of the colon may be relatively common in children.[3,8] Infants and children normally have hyperplastic lymphatic tissue, and further proliferation may be promoted by a variety of nonspecific stimulus such as infection or "allergy."[3,8]

Rabinowitz *et al.*[20] and Krasna and his associates[15] demonstrated lymphoid hyperplasia of the colon to follow the syndrome of necrotizing enterocolitis of the newborn in the absence of Hirschsprung's disease. The roentgenographic and pathologic features of lymphoid hyperplasia in our group of patients are identical to those described by the above authors.[15,20] It is, therefore, quite likely that lymphoid proliferation of the colon is a nonspecific sequel to an acute inflammation such as a nonspecific enterocolitis. In our small group of patients, however, there is no consistent correlation be-

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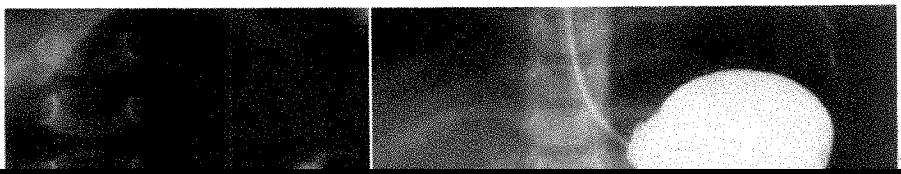

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ROENTGENOLOGIC AND CLINICAL MANIFESTATIONS OF NEONATAL NECROTIZING ENTEROCOLITIS*

EXPERIENCE WITH 43 CASES

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NEONATAL necrotizing enterocolitis (NEC) is an increasingly recognized and highly specific disease, first convincingly characterized in 1964 by Berdon and co-workers at Babies Hospital in New York.[1,12] Its clinical and roentgenologic features have recently been further defined by Touloukian *et al.*[21] in the same center, by Wilson and Woolley[23] of Los Angeles, and through previous communications from our institution.[16,17]

This paper presents the largest series yet reported and emphasizes the important role of radiology in establishing the diagnosis and in evaluating the severity and extent of the disease, which may affect as many as 1 out of 25 premature infants.

CASE MATERIAL

The medical records and roentgenograms were reviewed on all infants who had a surgical, pathologic, and/or roentgenologic diagnosis of NEC during the past 8 years at the University of Washington Affiliated Hospitals. Forty-three cases were identified, with 35 seen between January, 1968, and March, 1970. During this 27 month interval, the incidence was 3.7 per cent (24 newborns) of 651 admissions to the University Hospital Premature Center. This apparent recent "epidemic" is doubtless partially related to better recognition and to a lesser degree to slightly enhanced initial survival of susceptible prematures. Whether etiologic factors are becoming more prevalent is unknown at this time, but such is certainly suspected.

CLINICAL FINDINGS

MATERNAL HISTORY

Because many cases were referred to these hospitals, clinical information was not always complete. Maternal age was 16–20 years in 12 cases, 21–25 years in 13, 26–30 years in 7, and above 30 years in 6. For 14 mothers it was the first pregnancy, for 7 it was the second, for 10 the third, for 4 the fourth, and for 7 the fifth or more. Maternal blood groups were Type O in 11 patients, Type A in 11, Type B in 5, and Type AB in 1; 26 were Rh+ and 10 Rh−. The length of labor was 2–6 hours in 11 individuals, 6–12 hours in 13, 12–24 hours in 11, and more than 24 hours in 3. Four patients had cesarean sections. There were 7 instances of breech delivery, 1 case of cord prolapse, and 1 infant was delivered with the cord around his neck 3 times. Meconium staining of amniotic fluid was noted on 3 occasions.

Only 5 mothers could be described as having a completely uncomplicated prenatal history. Of the other 38, 13 had premature rupture of the membranes, 4 were followed for Rh incompatibility and induced at 34–36 weeks, 4 had urinary tract infections, 4 suffered from influenza or bronchitis, and 1 each had such various problems as diabetes mellitus, bicornuate uterus, hepatitis and rheumatoid arthritis with steroid therapy, cholestatic jaundice, leukemia treated with 5 fluorouracil, sickle cell trait with anemia, preeclampsia, mental retardation, hydramnios, diarrhea, ethanol

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Table I

SUMMARY OF CASES

Birthweight (grams)	No. of Patients	Gestational Age (weeks)	No. of Patients
800–1,200	12	less than 24	none
1,200–1,600	10	25–26	2
1,600–2,000	8	27–28	5
2,000–2,400	2	29–30	5
2,400–2,800	4	31–32	11
2,800–3,200	4	33–34	7
over 3,200	3	35–36	6
		37–38	2
		over 39	5

therapy for premature labor, and surgery for cervical incompetence. Many of these conditions are known to be associated with subsequent premature birth, and any relationship to development of NEC in the offspring is not yet known.

NEONATAL HISTORY

There were 23 males and 20 females in our series, whereas Touloukian *et al.*,[21] and Wilson and Woolley[23] had 7 males, 18 females and 10 males, 6 females, respectively. Thirty-six were Caucasian, 5 Negro, 1 Oriental, and 1 Indian-American. Touloukian *et al.*[21] noted a 60 per cent incidence of Negroes (probably related to hospital population); Wilson and Woolley[23] related a 25 per cent incidence. No significant increase in the occurrence of NEC was apparent at any specific time of the year.

Birthweights and gestational ages are given in Table I. Of our patients, 78 per cent were premature by weight (less than 2,500 grams), compared to 72 per cent of Touloukian *et al.*[21] and 25 per cent of Wilson and Woolley.[23] Gestational age was 36 weeks or less in 84 per cent. Many infants in the lower age and birthweight categories probably die before they have the opportunity to develop NEC, while older and heavier infants less frequently contract the disease or have milder cases which may go undetected.

Apgar scores were recorded as summarized in Table II. Touloukian *et al.*[21] reported that 7 of the 8 recorded scores in their patients were less than 8. Nearly 50 per cent of our series had Apgar scores of 6 or below, a high incidence of perinatal distress.

When first examined by the pediatrician, the majority had initial pulses in the 125–175 per minute range (35 of 43 patients) and respirations were 40–80 per minute (37 of 43). Acrocyanosis was a feature in 13, and in 3 the cyanosis was widespread (Touloukian *et al.*[21] reported cyanosis in 18 of 25 cases). In all 32 instances where it was mentioned, the umbilical cord had 3 vessels. Blood groups were as follows: 12 Type O, 13 Type A, 2 Type B, and 1 Type AB; 23 were Rh+, 5 were Rh−.

Of the 43 infants, 19 had evidence for the idiopathic respiratory distress syndrome (IRDS, hyaline membrane disease). Fourteen were mild cases and 5 moderate to severe. Most had abnormal blood gas analysis (32 of 43), and many (27 of 43) needed supplemental oxygen. The majority also manifested other significant diseases; *i.e.*, 6 had idiopathic jaundice, 3 had erythroblastosis fetalis requiring exchange transfusions, 3 had subarachnoid intracranial hemorrhage, and 1 each had hemophilia, cytomegalic inclusion disease (probably congenital), renal tubular acidosis, omphalitis, left-to-right shunt through an atrioseptal defect (ASD), ventriculoseptal defect (VSD), and patent ductus arteriosus (PDA), urinary tract infection, bladder distention, esophageal atresia with tracheoesophageal fistula, aspiration pneumonia, a left renal cyst, neonatal chronic pulmonary disease, diarrhea and thrombocytopenia.

Table II

APGAR SCORES

	At 1 minute	At 5 minutes
0	1	0
1–2	6	1
3–4	5	2
5–6	7	7
7–8	13	5
9–10	8	9
not done	3	19

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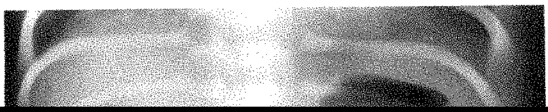

5. *Abnormal barium enema examination.* Due to the virtually pathognomonic plain film roentgenographic findings and the fre-

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Table III

INCIDENCE OF ROENTGENOGRAPHIC FINDINGS

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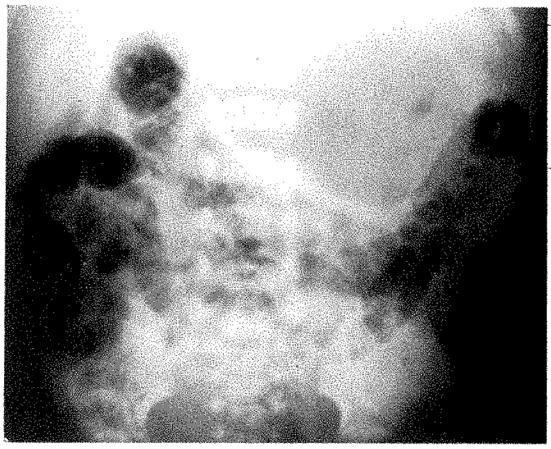

FIG. 1C. Total colonic involvement with cystic pneumatosis intestinalis, demonstrating submucosal

feasible. Details of the surgical treatment have been discussed by this group previously.[16,17] Fifty-five to 60 per cent of both the series of Touloukian *et al.*,[21] and Wilson and Woolley[23] required surgery.

PATHOLOGY

Involvement is usually centered around the terminal ileum, cecum, and right colon. Of the 28 cases with surgical or autopsy proof, the regions affected were as shown in Table IV. Some degree of colonic involvement was present in 25 of 28 cases. Isolated involvement of proximal small bowel (1 case), terminal ileum (2 cases), or sigmoid colon (1 case) is unusual, with stomach,

[redacted]

extensive subarachnoid or intracerebral

TABLE V

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Treatment consists of supportive measures, wide-spectrum antibiotics, and surgery (52 per cent of this series), with a combined mortality of 37 per cent. Long term sequelae are rare.

The etiology is unknown. It is hypothesized that a primitive reflex redistribution of splanchnic blood flow, in response to significant infant stress, results in bowel ischemia with subsequent tissue damage and secondary bacterial invasion.

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VOL. 112, NO. 1

PROBLEMS WITH MECONIUM*

By ARTHUR S. TUCKER, M.D., *and* ROBERT J. IZANT, JR., M.D.
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THE word *meconium* appears in the names of 4 distinct disorders. The purpose of this paper is to discuss and differentiate these 4 entities, which are: (1) meconium peritonitis; (2) meconium ileus; (3) meconium plug syndrome; and (4) meconium-ileus equivalent (kotileus).

The first three of these are encountered in the newborn period. The fourth is a later complication of cystic fibrosis. The designation meconium-ileus equivalent is, in a way, misleading. Meconium is not present at all, because it is never found in children more than a few days old. Jensen applied the name to the mechanical bowel obstruction sometimes encountered in patients with cystic fibrosis who have thick putty-like stool in their intestines.[19] Because of the possibility of confusion with the other disorders involving problems with true meconium, we suggest referring to this condition by its original German name of kotileus.[32]

THE NATURE OF MECONIUM

Meconium is not a disease. Meconium is a normal component of the newborn infant's intestinal tract. The term is derived (through Latin) from the Greek *meconarion*, "from the poppy." Perhaps this reflects a belief of the ancients that it was a substance (similar to opium) which kept the fetus asleep and comfortable in the uterus. In any event, it is a name dignified by usage since Aristotle.[14]

The meconium found in the intestine of the normal newborn is a large mass of mucous material containing mucoprotein and mucopolysaccharides. Like most other body substances, it is largely (75–85 per cent) composed of water. Biliverdin passed down from the liver gives it the characteristic dark green color. Histologic examination shows it to contain "meconium bodies," which are keratinized and nucleated squames shed from the skin into the amniotic fluid and swallowed by the fetus. As long ago as Hippocrates it was recognized that "The presence of excretion in the intestine of the newborn baby proves that the foetus sucks inside the womb."[14]

Meconium causes no difficulty as long as it is eliminated from the bowel during the first day or two of life. If it remains longer in the infant, it complicates his life.

MECONIUM PERITONITIS

Among the misadventures which may befall the child during his intrauterine development is the first of 3 meconium afflictions that are encountered in the newborn, *viz.* meconium peritonitis. Meconium peritonitis consists of an inflammatory reaction in the peritoneal cavity of the newborn which apparently always is a sequel of intestinal perforation *in utero*. A sterile abscess of varying extent results from exposure of the serous surfaces to the irritating material. In the process of healing, calcification takes place rather rapidly in a variable distribution (Fig. 1*A*). The calcification can be seen microscopically probably in every case. It may take a few weeks, however, for calcification to become dense enough to be visible roentgenographically in the patient.

The origin of the original perforation may be a developmental defect or an acquired one secondary to volvulus, internal hernia, or intussusception, but we believe the overwhelming majority of perforations to be basically the result of an interruption in blood supply to the bowel, as docu-

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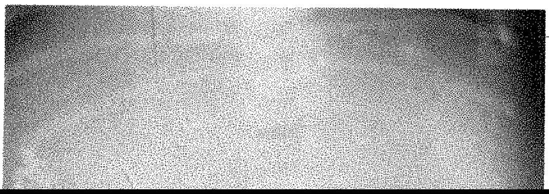

with the meconium peritonitis (as is usually the case), laparotomy is required to resect the dilated and hypertrophied proximal intestine and re-establish continuity of the remaining bowel.[29] Some intrauterine per-

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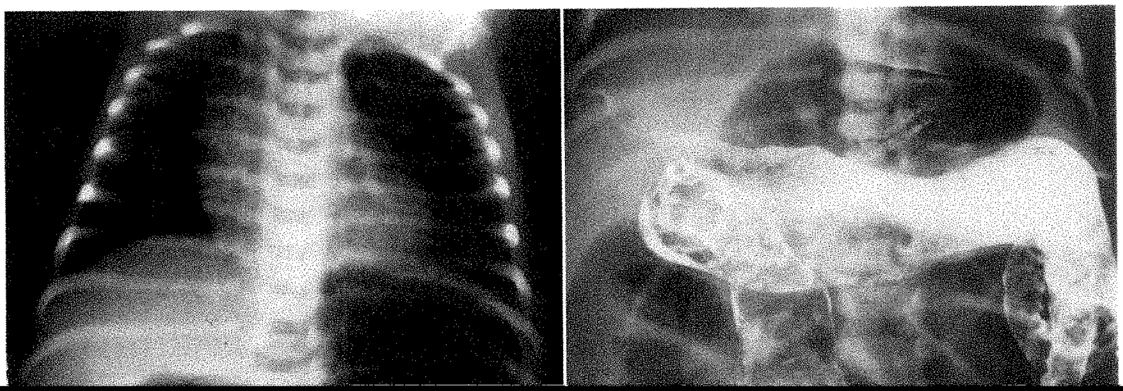

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difficulty after eliminating the meconium

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bowel movements and is able to palpate

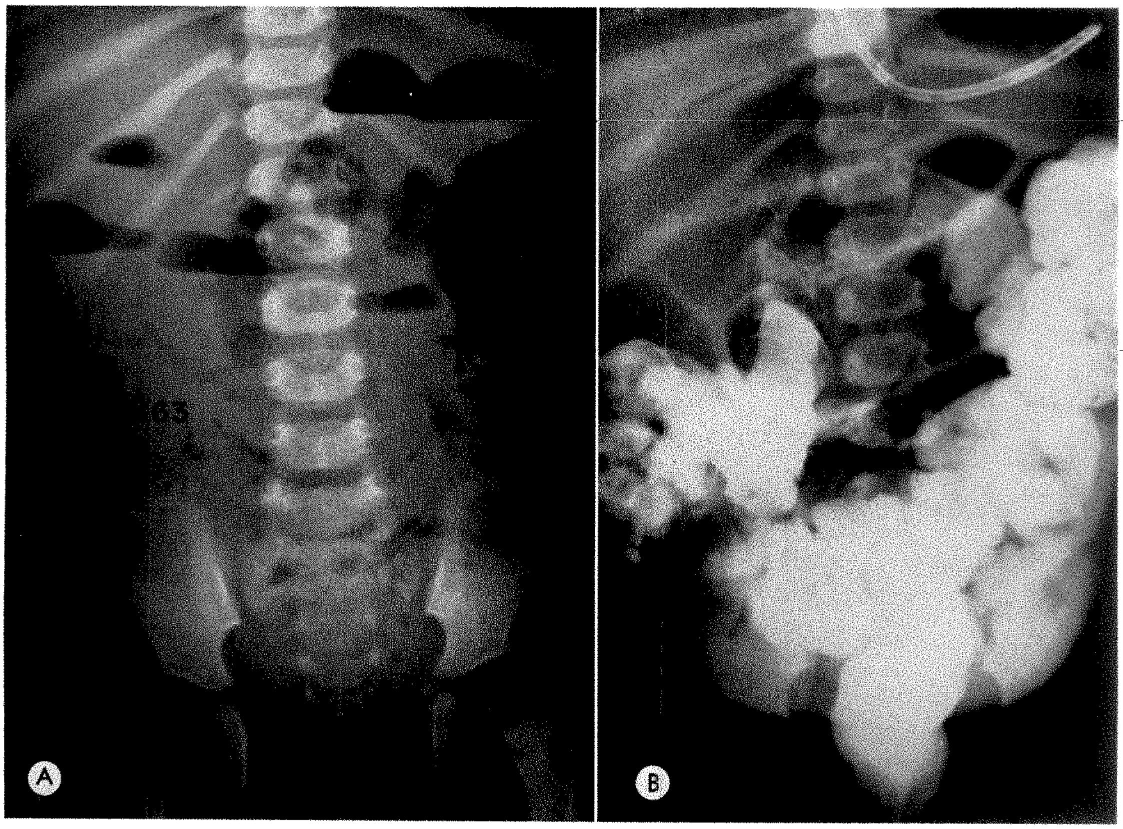

Fig. 5. *Meconium-ileus equivalent* in the same patient shown in Figure 2. At the age of 14 months the girl was admitted with a history of obstipation and anorexia for 2½ days and vomiting for 1 day. The child had not been receiving her pancreatic enzymes with regularity. Physical examination revealed an ill-defined, soft, indentable mass in the right lower quadrant.

(*A*) The erect roentgenogram of the abdomen shows small intestinal obstruction, with evidence of air admixed with much stool in the transverse colon. The patient was treated with enemas, and cotazym capsules administered by nasogastric tube. (*B*) The next day, a gastrografin enema filled the colon and entered the dilated terminal ileum. Following this, the patient expelled a very large, pale, foul stool, and her abdominal distention subsided.

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EXTRAHEPATIC PORTAL OBSTRUCTION IN CHILDHOOD AND ITS ANGIOGRAPHIC DIAGNOSIS*

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EXTRAHEPATIC portal obstruction in children is usually due to occlusion of the portal vein, either by a congenital anomaly or as an abnormal consequence of the postnatal cessation of placental circulation.[1,4] In the latter case, the process responsible for obliterating the fetal umbilical vein and ductus venosus (Arantii) extends abnormally to involve the portal system. In addition, omphalitis, other abdominal inflammation, sepsis, exchange transfusion and acute dehydration can cause portal vein obstruction in children.

PATHOLOGY

By causing extrahepatic portal hypertension, portal vein occlusion leads to splenomegaly, sometimes mild, but always present,[5] and collateral blood flow of two types; *i.e.*, hepatopetal to the liver, and hepatofugal, away from it. Hepatopetal collaterals, often extensive, carry blood to the undamaged liver, functionally replacing the portal vein. In the form of complex networks surrounding the obstructed portal vein, within the hepatoduodenal and hepatocolic ligaments, around the gallbladder, and over the surface of the liver, these hepatopetal collaterals terminate in middle-sized intrahepatic portal veins and collectively, if loosely, are referred to by the term, "cavernomatous transformation of the portal vein."

Hepatofugal collaterals develop due to insufficiency of hepatopetal collaterals and serve to shunt part of the blood normally carried by the portal vein into the inferior and superior vena caval systems. They are formed in areas of normal interconnection between the portal and caval circulations, including the distal esophagus, the cardia, rectum, umbilicus, mesentery and the retroperitoneal space. Similar collaterals also form in postinflammatory adhesions around the abdominal viscera, especially the spleen. Direct communications between the splenic vein and the inferior vena cava or left renal vein may also develop. Hepatofugal collaterals are usually large and often form varices.

SYMPTOMS

Clinically, extrahepatic portal obstruction presents at various ages with the various manifestations of Banti syndrome.[2,6,8] In patients with postomphalic pyelothrombosis, it usually appears early (average age of onset 3 years); when it follows general infections or is idiopathic, clinical manifestations are delayed sometimes even into adolescence (average age of onset 8 years).[8] The sudden onset of severe gastrointestinal bleeding with hematemesis may be an initial manifestation. Some children have chronic gastrointestinal bleeding with anemia or symptoms of hypersplenism; some have chronic diarrhea; in others, distended abdominal and thoracic veins are present. Incidentally discovered splenomegaly may be the only manifestation. Ascites and jaundice do not occur in children with extrahepatic portal obstruction.

DIAGNOSIS

Blood and liver tests provide useful information in the differential diagnosis of childhood portal obstruction. Barium studies may aid by revealing esophageal and gastric varices. The definitive anatomic diagnosis is provided by angiography.

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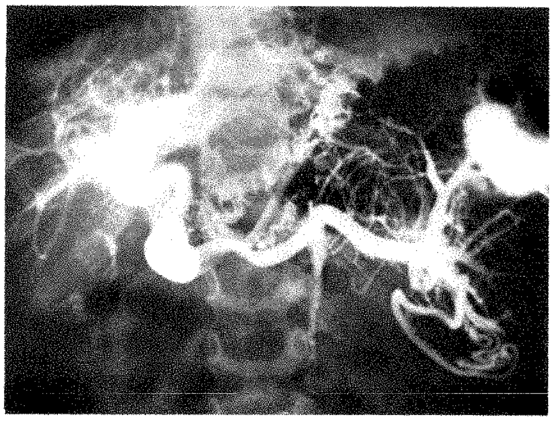


Fig. 1. *Portal vein occlusion with extensive hepatopetal and hepatofugal collateral circulation.* A 3 year old child with negative perinatal ~~history and spleno-~~

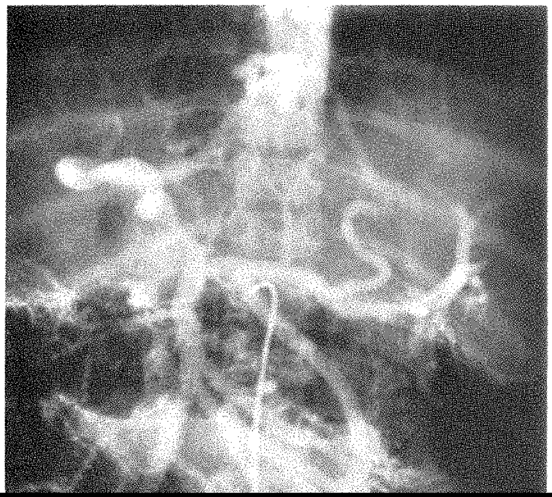
~~ranged from 2 to 18 years and averaged 7.2~~ years. With respect to etiology, 12 had perinatal infections, usually omphalitis; 3 had received exchange transfusions; and 1 had postnatal surgery for congenital anomaly of the bile ducts. The other 22 cases were idiopathic. Splenomegaly was present in all patients and 20 had gastrointestinal bleeding.

Splenoportography was done in all but 5 patients, who had had previous splenectomy. Twenty patients were examined by celiac and superior mesenteric arteriography. In 2 cases, inferior mesenteric arteriography and in 2 cases, inferior vena cavography was also done.

[redacted]

Fig. 4. *Portal vein occlusion with poor hepatopetal but extensive hepatofugal collateral circulation.* A 4 year old child with a history of postnatal exchange transfusions and recent gastrointestinal bleeding.

Venous phase of superior mesenteric angiography. The portal vein is replaced by a large, tortuous collateral running from the hepatic hilum to the lower esophagus and supplying esophageal varices. There is retrograde flow to the enlarged inferior mesenteric vein and splenic vein with filling of gastric and perisplenic veins.



The portal vein was completely occluded in 37 of the 38 patients in this series

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splenomegaly, the portal vein was normal but its right hepatic branch and the trunk of the superior mesenteric vein were both occluded (Fig. 7, *A* and *B*).

Hepatopetal collateral circulation of varied appearance was present in all patients. In some, one or two large, tortuous veins originated from the portal or splenic vein proximal to the site of obstruction and ran to the hepatic hilum to enter a plexus of small collaterals (Fig. 1). More often, however, a number of small or middle-sized and usually tortuous collaterals filled, forming an extensive collateral network in the subhepatic region (Fig. 2; and 3). The middle-sized and small intrahepatic vessels which filled from this network were of normal appearance, excluding an intrahepatic process. There was no filling of major intrahepatic portal branches.

Hepatofugal collaterals were present in all but 1 of our patients, a 4 year old girl with

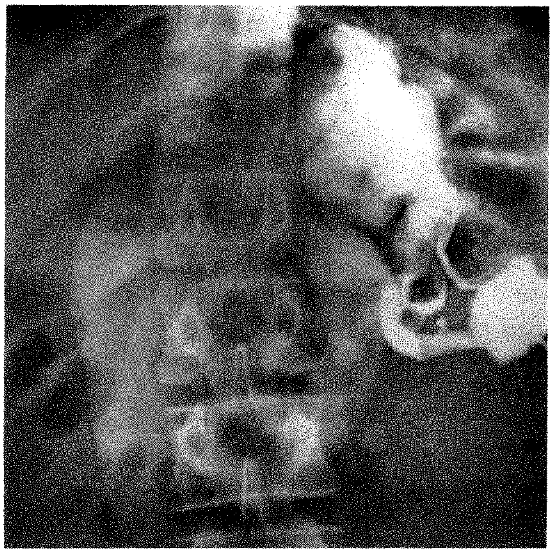

Fig. 6. *Portal vein occlusion with extensive hepatofugal collaterals and spontaneous splenocaval shunt.* A 14 year old child without any positive history and splenomegaly as the only finding.

Splenoportogram. Contrast medium injected

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splenic region, retroperitoneal space and, in lesser degree, the intestine. Enlarged gastric coronary and short gastric veins formed varices in the upper part of the stomach, cardia and esophagus, variable in degree and quite extensive in some cases (Fig. 1–5).

The perisplenic collaterals were mostly small and entered veins in the retroperitoneum and abdominal or thoracic wall. In 5 patients, greatly dilated, variceal retroperitoneal channels carried all the contrast medium injected into the spleen either into the left renal vein (Fig. 5, *A* and *B*) or over the spine and directly into the inferior vena cava (Fig. 6).

Portal obstruction was accompanied by arterial anomalies in 5 of the 20 patients

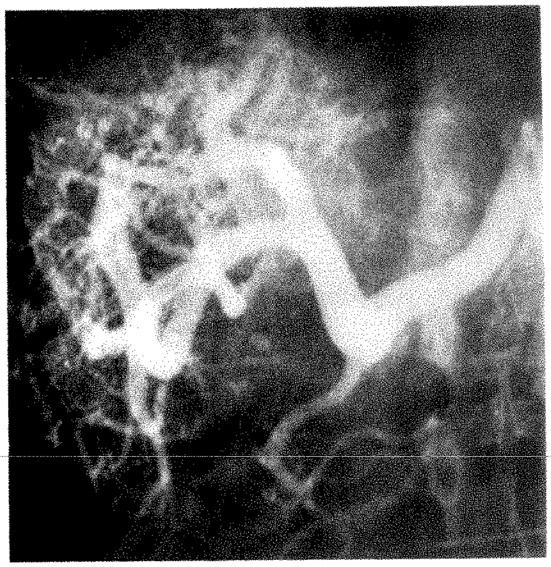

FIG. 9. *Angiomatoid hypertrophy of the arterial supply*

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MESENTERIC CYST ASSOCIATED WITH PROTEIN LOSS IN THE GASTROINTESTINAL TRACT*

STUDY WITH LYMPHANGIOGRAPHY

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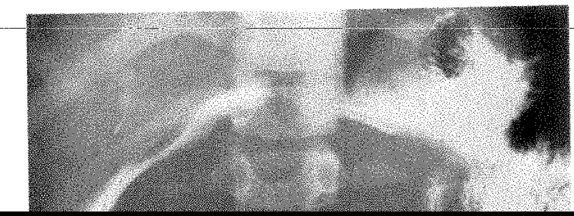
CYSTIC lymphangiomas of the mesentery and omentum (also called chylangiomas, chylous cysts, mesenteric and omental cysts) are benign, unilocular or multilocular, endothelium-lined cystic tumors, containing serous or chylous [redacted]

significant abdominal distention with questionable shifting dullness. Physical examination was otherwise noncontributory. No abdominal masses were palpated. Hemoglobin was 9.8 gm. per 100 ml., hematocrit 34 per cent, and white blood cell count 4,900 per ml. A sickle cell preparation was negative. Urine was clear and tionship of the mass with the blood supply of the bowel, surgical excision was not feasible. The mass was biopsied and drained. A large amount of chyle was obtained, and copious amounts of chylous fluid drained for the next several weeks.



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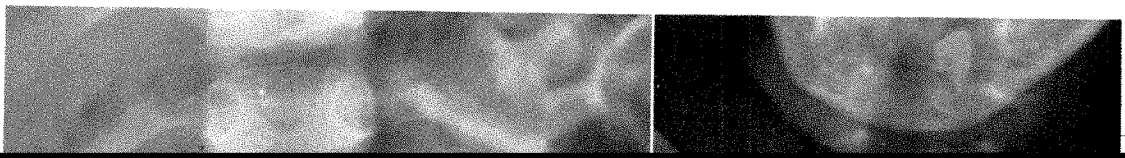

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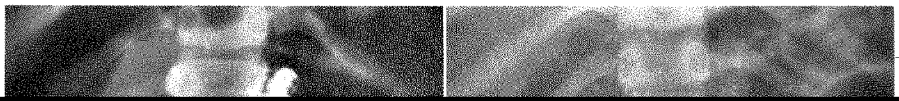

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TERATOMAS IN INFANTS AND CHILDREN*

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TERATOMAS are true tumors of congenital origin containing multiple tissues foreign to the part in which they are located. Excluded from this definition are heterotopic tissues such as epidermoid inclusion cysts and dermoid inclusion cysts beneath the skin or in the cranial cavity and forms of suppressed twins.[52,53]

Although the over-all incidence of teratoma is somewhat rare, the proportion of cases discovered in infancy and childhood is significant. In Willis' series, 62 of the 172 teratomas occurred in children less than 16 years of age.[53]

A consideration of the various presentations and roentgenographic features of these tumors forms the basis of this paper. In the period from 1949 to 1970, 42 cases of teratoma were diagnosed at the Children's Hospital Medical Center in patients under 16 years of age (Table I).

Table I

INCIDENCE OF TERATOMA BY SITE

	Total	Malignant
Ovarian	15	3
Sacrococcygeal	12	4
Testicular	4	—
Nasal-nasopharyngeal	3	—
Retroperitoneal	2	1
Cervical	2	—
Mediastinal	2	—
Intracranial	2	2
	42	10

REPORT OF CASES

Eight representative cases have been chosen to be presented here in some detail:

Case I. *Intracranial teratoma of the newborn.* D.L.S. This 7 week old female infant, born on January 18, 1969, was admitted to the hospital on March 11, 1969 with the diagnosis of "hydrocephalus." The parents had consulted a physician because the infant's eye had appeared unnatural during the week prior to the admission. On physical examination, the head circumference was 41 cm., the fontanelle was tense, and a "setting sun" appearance of the eyes was present.

Intracranial calcification was not detected on the plain film roentgenograms. The ventriculographic examination showed dilatation of the lateral ventricles. Within the lateral ventricles a large, round mass with an irregular surface was outlined (Fig. 1, *A* and *B*). The third ventricle could not be filled with air despite various positional maneuvers of the head. A shunt procedure between the right lateral ventricle and the right atrium of the heart was performed on March 17, 1969. The infant's head continued to expand to 45.5 cm. over the following 2 week period. A bilateral cerebral angiography was performed prior to craniotomy. The angiographic manifestations of hydrocephalus were noted. Shift of the anterior cerebral arteries to the left of midline associated with axial rotation of the posterior cerebral arteries suggested gross distortion of the central and deep brain structures by a tumor mass (Fig. 1, *C* and *D*). Abnormal vessels, consistent with a malignant intraventricular tumor, emanated from the terminal distribution of enlarged choroid arteries. In the late arterial phase, early venous shunting in this region was seen. An intense vascular blush in the tumor mass was not detected.

Craniotomy revealed a brown, firm, intraventricular mass on the right, extending from the frontal horn anteriorly to the atrium posteriorly. It could not be ascertained at surgery whether the tumor was obstructing the foramen of Monro. Only a biopsy specimen was obtained, since the tumor was too extensive to

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Bilateral mild papilledema and bitemporal field defect were present. Cerebral arteriography revealed stretching of the carotid arteries just above the siphons. ~~The pneumo-~~encephalographic examination demonstrated a suprasellar mass impinging upon the third ventricle anteroinferiorly (Fig. 2). At the surgical exploration a pale yellowish-pink midline mass located between the two optic nerves was found. On the external examination, the mass was quite firm. The tumor was totally removed. The fragment removed for frozen section study measured 1.4×0.8×0.7 cm. in maximum dimensions and the remaining portion of the tumor measured 3.0×1.5×1.1 cm. in maximum dimensions. The histologic study of the 2 fragments was reported as representing malignant teratoma. The well differentiated components consisted of various sized cystic spaces; were lined by goblet cells containing intestinal type epithelium; and some were lined by cuboidal epithelium. Occasional plate of hyaline cartilage was seen. Also found were adipose tissue, hair follicles with hair shafts, sebaceous glands, sweat ductules, nests of sweat glands, nerve segments, mucous glands, pancreatic tissue, smooth muscle and fibrous connective tissue. Some extremely primitive mesenchymal tissue and completely undifferentiated cells were found in the tumor.

Comment. The above described 2 cases reflect what has been reported in the literature. The intracranial teratomas in the newborn are usually large and cause death at birth or within a few weeks after birth. On the other hand the tumors in the older children are smaller in size with the symptoms developing gradually over a relatively long period of time.

Case iii. *Nasal teratoma.*

M.C.L., a female infant, was admitted to the hospital on January 3, 1955 at 9 days of age because of a midline tumor of the nasofacial area. She had required gavage feeding since birth and was noted to have cyanosis during crying episodes. A firm tumor attached to the mid-anterior palate and nose was present. The surface of the tumor was somewhat irregular with three fleshy appendages attached to the lower aspect of the mass. The mass did not transilluminate.

Plain film roentgenograms of the mass demonstrated several calcific densities that showed ~~some evidence of osseous organization. Anter-~~iorly, it split the maxillae and nasal bones. A small calcific density was also noted intracranially in the midline (Fig. 3, *A* and *B*). In the pneumoencephalographic examination intracranial extension of the mass was not recognized. The intracranial calcification was located superior to the cavum septi pellucidi. At surgery the nasal bones were found to be separated laterally by the mass. The base of the mass was dissected free from the maxilla laterally and the vomer in the midline and removed completely. The tumor extended to the apex of the nose but did not enter the cranial cavity. The specimen measured 5×4×4 cm. in size. Microscopic sections of the mass showed areas of immature fat and numerous collections of mucus glands. Several fragments of cartilage and bone with characteristic bone marrow were noted. The final diagnosis was benign midline teratoma. At 8 years of age the child showed no evidence of neurologic or intellectual impairment. Several restoration procedures have given cosmetically satisfactory results.

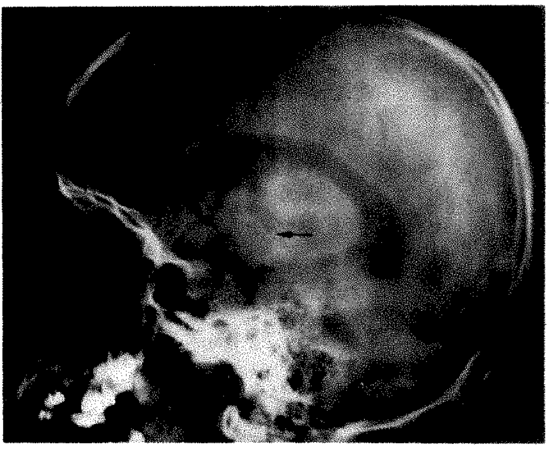
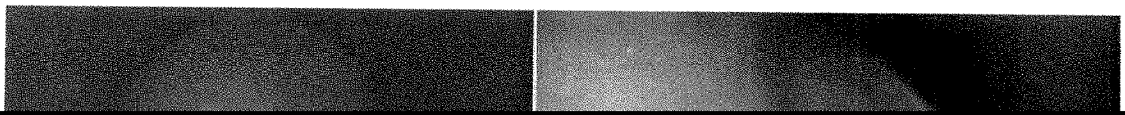


Fig. 2. Case ii. *Suprasellar teratoma.* Pneumoencephalographic examination shows a suprasellar mass impinging upon the third ventricular space (arrows).

Comment. The significance of the intracranial calcification is uncertain. A small teratoma within the skull remains a possibility. Some nasopharyngeal teratomas have a direct extension into the cranial cavity.[56] This was not found in the present case.

Case iv. *Mediastinal teratoma.*

C.W.R. This 7 year old male patient was admitted to the hospital on January 5, 1967. ~~He complained of mild pain over the manu-~~brium of 10 days' duration. The pain was dull



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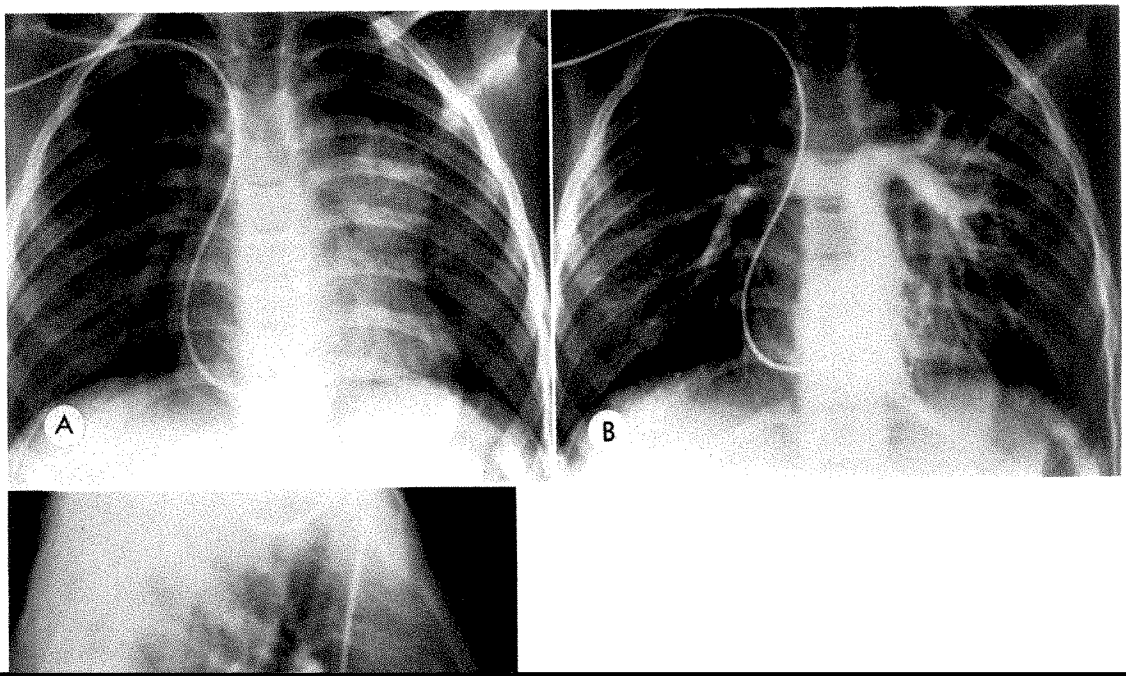

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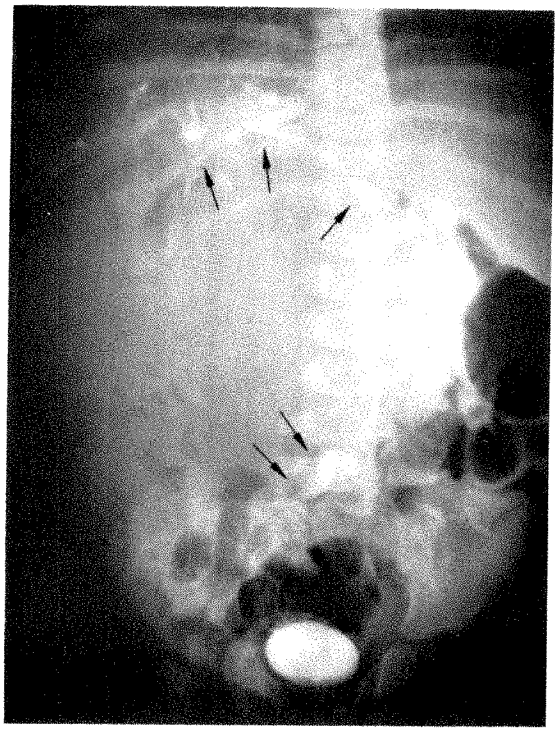


FIG. 5. Case V. *Retroperitoneal teratoma.* Excretory urography and total body opacification show a large and relatively radiolucent mass in the right abdomen with extension to the left of the midline. The right kidney is displaced downward and to the left (lower arrows). Bands and streaks of calcium are seen in the upper abdomen (upper arrows).

ependyma, immature connective tissue, bronchial mucosa and hyaline and elastic cartilage were found in the tumor.

Comment. This is an unusually young age for ovarian teratoma to be symptomatic in a child. These tumors were diagnosed between 3 to 14 years of age in the remaining 14 cases included in this study. The spillage of the tumor into the peritoneal cavity and implantation on the visceral and parietal peritoneum are other interesting features of this case.

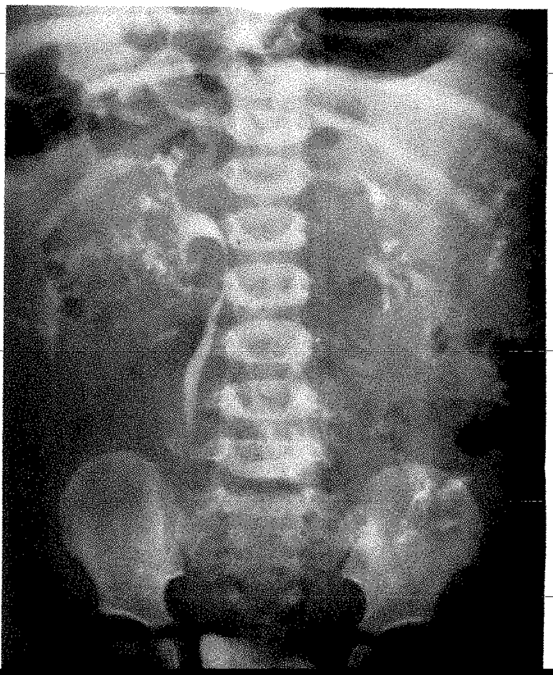
Calcification was found on the abdominal roentgenograms in 4 of the patients in this series. Extrinsic deformity of the bladder was present in 3 cases and of the bowel in 4 cases.

CASE VII. *Benign cystic teratoma of the testicle.* J.K. This 7 year old male was admitted to [redacted]

angular calcific density approximately 7 mm. in length in the right scrotal region (Fig. 7). The right testicle was removed on September 15, 1969. The testicle measured 3.0×2.5×1.5 cm. The specimen was that of a benign multicystic teratoma with normal appearing residual immature testis, epididymis, rete testis, and tunica albuginea. Glial tissue with foci of choroid plexus, a few scattered neurons and somewhat irregular, heavily pigmented epithelium consistent with an optic cup were found in the mass. The multiple varying sized cysts were lined variously by cuboidal epithelium, stratified squamous epithelium and ciliated pseudostratified columnar epithelium of the respiratory type. Calcified material in the configuration of the tooth buds was also present.

Comment. In one other testicular teratoma in this series, calcification was present in the tumor. Presence of calcification on the roentgen study is of help to differentiate this tumor from the others, in particular embryonal cell carcinoma.

CASE VIII. *"Tail-like" sacrococcygeal teratoma.* T.McC. This newborn female infant was



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ovary is grouped together with teratomas. Hair, teeth and nervous tissue arise from

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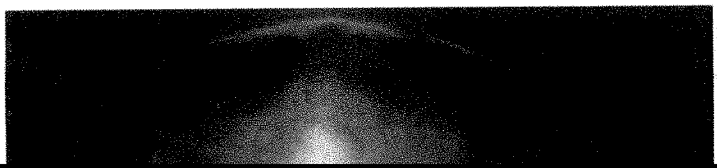
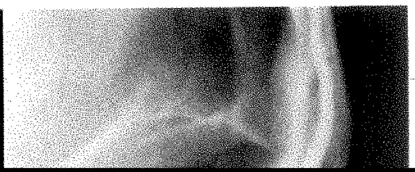
with less severe enlargement of the head. Death occurred within 9 weeks and was





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since it is the most common type of ovarian neoplasm in this age group and has the capacity for malignant change. The cystic form of the tumor is most common and is usually benign. The solid teratomas although less frequent are commonly found to be malignant.[16]

Clinically the symptoms may be related to: torsion, abdominal mass, precocious sex change, or mechanical effect on surrounding viscera.[34] Torsion may simulate appendicitis. This occurred in 3 patients in this series. Unlike appendicitis, the acute symptoms are generally preceded by similar but less severe episodes in the past.[7] In some cases serial development of teeth has been recorded.[57]

TESTICULAR TERATOMAS

Most tumors of the testes are detected during the first 3 years of life.[3,8] The most common malignant tumor during that period is the embryonal cell carcinoma.[49] The teratoma is the most common benign testicular tumor of childhood. Malignant degeneration of the teratoma is quite rare in early childhood, but becomes more frequent after adolescence. The diagnosis of testicular teratoma can often be suggested by the demonstration of calcification within the tumor. This diagnosis has clinical significance since the treatment of teratoma is by simple surgical extirpation of the tumor which usually results in cure. Embryonal cell carcinoma which does not calcify requires more extensive surgery and is associated with a far less favorable prognosis.

SACROCOCCYGEAL TERATOMA

A review of the literature by Hunt *et al.*, includes 202 cases of this tumor.[25] They occur predominantly in girls with a frequency of 70 to 90 per cent. Thirteen of 111 cases reviewed by Waldhausen *et al.* were associated with dystocia and their delivery required cesarean section.[50] Most of the tumors are attached to the coccyx and protrude externally above the gluteal crease. Less often the tumor may extend retroperitoneally into the pelvis and rarely into the spinal cord.[17] Calcification is detectable on the roentgenogram in about one-half of the cases.[17,18,32,56] This feature is helpful in differentiating the tumor from meningocele. In addition, the teratoma usually differs from the meningocele in that it does not deform the sacrum.[51] Exceptions are malignant teratomas which may cause sacral erosion.[51] Destruction of the coccyx may be seen with either benign or malignant teratoma. The rectum frequently is deformed posteriorly from the presacral extension of the mass. Less frequently complete obstruction of the rectum and ureters may occur.[50] In 1 patient the tumor has been cited in association with hydrometrocolpos.[19]

Malignant degeneration is one of the most important considerations in the early diagnosis and treatment of sacrococcygeal teratoma. Dramatic changes in the incidence of malignancy have been noted in the review of the literature:[25] 6.5 per cent from birth to 4 months; 50 per cent from 5 months to 5 years; 5 per cent over 5 years of age.

A condition to be considered in the differential diagnosis is that of vestigial tail.[22,28,35,54] This entity should be distinguished from true teratoma since it is not known to undergo malignant change. The tail may even demonstrate movement.[22,35] An occult tail is enclosed in the buttocks, but fully developed vertebrae may be seen by roentgen examination.[43]

SUMMARY

The clinical and roentgen manifestations of 42 cases of teratoma have been reviewed.

Eight of these are described in some detail.

A brief review of the literature on this subject is presented.

The importance of early diagnosis is stressed because of the potential for malignant change and the compromise of vital structures by compression or invasion.

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The authors would like to express their appreciation to Dr. Peter Kane for his interpretation of the contrast studies of Cases I and II, and to Dr. Fred Sondheimer for his evaluation of Case I. Our thanks are due to Mrs. Marilyn Merrell, Mrs. Ruth Mainzer and Miss Joy Reeves, for their secretarial work and to Mr. William Law for his technical assistance.

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ARTERIAL STENOSES IN NEUROBLASTOMA*

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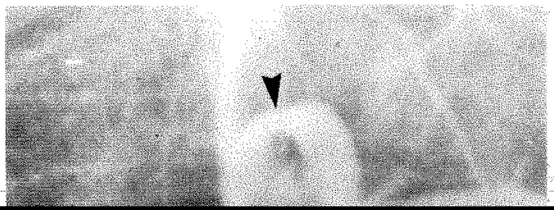

NEUROBLASTOMA is the second most common abdominal neoplasm of childhood. There have been sporadic reports of the use of angiography in this condition,[1,2,4] and an account of the arteriographic findings in 13 patients has been published.[3]

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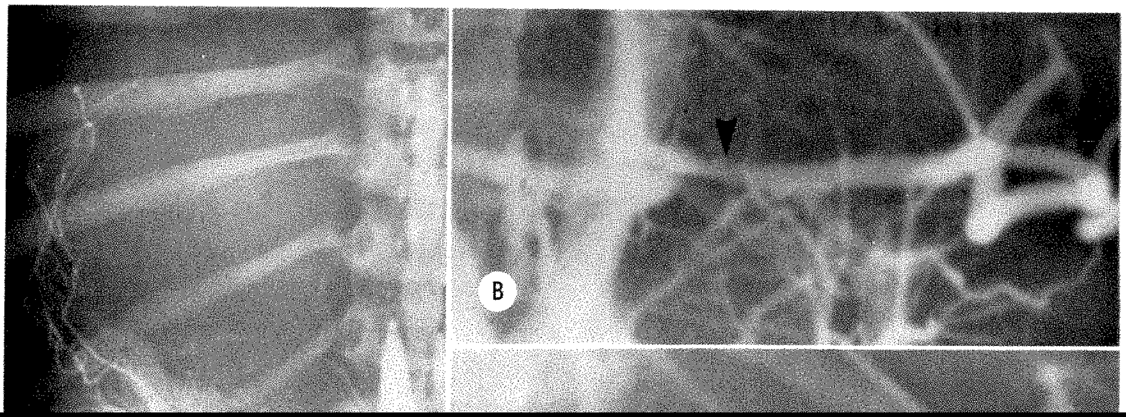

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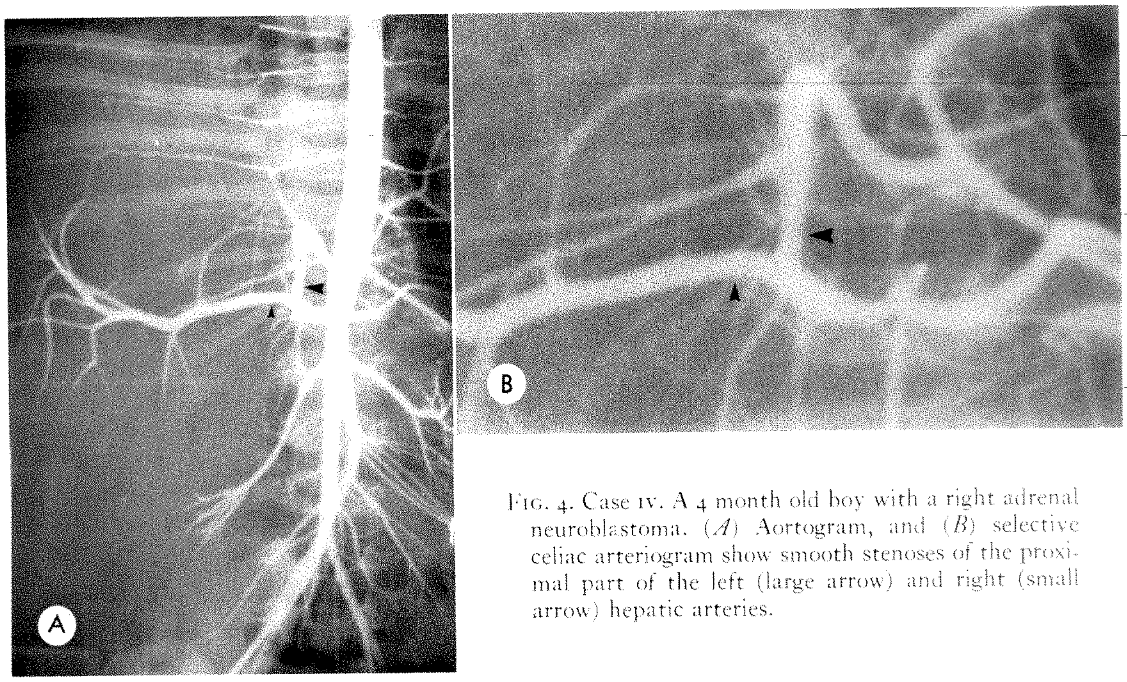

Fig. 4. Case iv. A 4 month old boy with a right adrenal neuroblastoma. (*A*) Aortogram, and (*B*) selective celiac arteriogram show smooth stenoses of the proximal part of the left (large arrow) and right (small arrow) hepatic arteries.

an important part of this evaluation and

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MEGAURETER IN INFANTS AND CHILDREN: ROENTGENOLOGIC, CLINICAL, AND SURGICAL ASPECTS*

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MEGAURETER, a relatively common problem in pediatric urology, has received more attention in the past few years as a result of new surgical techniques. The term "megaureter" is used here to describe any markedly dilated ureter that is either secondary to obstruction at the ureterovesical junction or secondary to massive vesicoureteral reflux. Dilated ureters caused by ureteroceles or neurogenic bladder have not been included in this evaluation.

This paper is based on experience with operative correction of megaureter during the past 10 years by one of us (W.H.H.). Certain aspects of this work have been reported previously.[2–4] This study is based on the treatment of 71 ureters in 46 patients (30 boys, 16 girls). Obstruction was present in 24 distal ureters, and massive reflux in the remaining 47. We have not included several cases in which there was megaureter associated with a totally destroyed kidney which was treated by nephroureterectomy instead of reparative surgery.

In the past decade, various methods have been applied in the treatment of megaureter, including triple-voiding technique in refluxing megaureter;[7] burying the megaureter into adjacent psoas muscle; replacement of the megaureter with a segment of intestine;[8] and ureterostomy as a temporary or permanent diversion.[9] Bischoff[1] used an aggressive approach by reducing the caliber of the ureter, shortening it, and performing an osteoplasty to avert reflux. Paquin[6] also shortened and tapered the dilated ureter following preliminary nephrostomy. Johnston[5] advocated operative correction of megaureter. A review of the literature indicates that corrective surgery produces a favorable result for this condition.

In order to evaluate megaureter, the radiologist should be acquainted with the anatomy, pathology, radiologic findings both preoperatively and postoperatively, and the current surgical techniques that can be used to correct this serious problem.

CLINICAL FEATURES OF MEGAURETER

Megaureter can be either unilateral or bilateral. The dilatation may be secondary to obstruction (mechanical or functional) in the terminal ureter (Fig. 1*A*; 4*A*; and 5*A*), or it can result from massive reflux. In obstructive megaureter the caliber of the intravesical segment of the ureter may be narrow, but usually a ureteral catheter can be passed through it. A typical case of moderate severity is shown in Figure 4*A*. Resection of the narrow segment combined with tapering of the ureter for several centimeters and reimplantation into the bladder can give an excellent result, as shown in Figures 4, *A* and *B*, and 5, *A–D*. In the refluxing megaureter the intravesical tunnel is usually very short (less than 5 mm.), and the ureteral orifices in the bladder are frequently gaping and appear as "gopher holes" at cystoscopy. The dilated ureter shows hypertrophied muscle and sometimes considerable fibrous tissue when chronic infection has occurred (Fig. 1*C*). In some cases the ureter is quite elongated and tortuous, which is associated with kinking (Fig. 1*A*). This tortuosity appears to result in most cases from high-grade obstruction originating in intrauterine life, but in others

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Table I

ASSOCIATED ANOMALIES IN 46 PATIENTS WITH MEGAURETER

	No. of Patients
Prune Belly Syndrome	3

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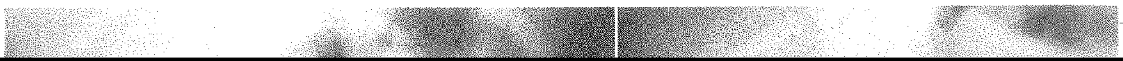

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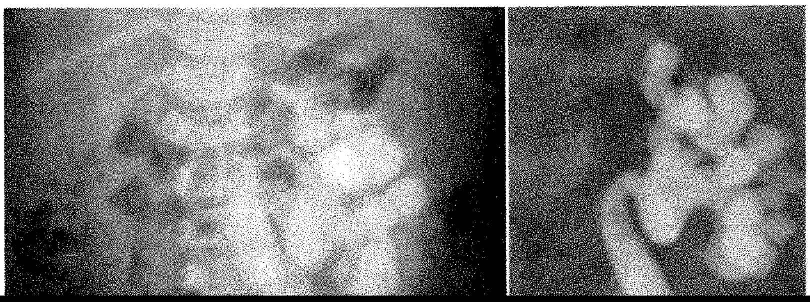

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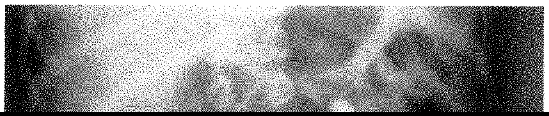

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TABLE IIA

ROENTGENOGRAPHIC FINDINGS OF THE HANDS AND WRISTS IN KLINEFELTER'S SYNDROME

Roentgenographic Findings	XXY	XXXY XXXXY	XXYY	Total
Metacarpal sign positive	3/13	0/2	0/2	3/17
Phalangeal preponderance	3/13	0/2	0/2	3/17
Fifth finger				
Brachymesophalangia with clinodactyly	2/13	1/2	1/2	4/17
Brachymesophalangia without clinodactyly	1/13	0/2	0/2	1/17
Simple clinodactyly	2/13	0/2	1/2	3/17
Flattened ulnar styloid process	1/13	0/2	0/2	1/17
Accessory epiphysis of second metacarpal	1/13	0/2	1/2	2/17

No. of positive findings/No. of observations.

of the skeletal system appeared normal in all cases.

The roentgenographic findings of the hands and wrists are summarized in Tables IIA and IIB.

The incidence of the borderline or positive metacarpal sign was reported as 13 per cent in 100 control subjects of various ages according to Kosowicz,[8] and approximately 8 per cent in the larger normal population studied by Archibald *et al.*[1] In 3 of 17 (17.7 per cent) cases of Klinefelter's syndrome observed by us this sign was positive (Fig. 1), whereas in 40 controls this sign was positive in 3 (7.5 per cent) and borderline in 2 (5 per cent).

TABLE IIB

ROENTGENOGRAPHIC FINDINGS OF THE HANDS AND WRISTS IN CONTROLS

(Japanese: age range 20–61 years)

Roentgenographic Findings	Male	Female	Total
Metacarpal sign			
Positive	2/20	1/20	3/40
Borderline	0/20	2/20	2/40
Phalangeal preponderance	12/20	12/20	24/40
Fifth finger			
Brachymesophalangia with clinodactyly	0/20	1/20	1/40
Brachymesophalangia without clinodactyly	1/20	1/20	2/40
Simple clinodactyly	0/20	1/20	1/40
Flattened ulnar styloid process	1/20	0/20	1/40

No. of positive findings/No. of observations.

Comparison of the length of the fourth metacarpal with the total length of the distal plus proximal phalanges of the fourth finger was said to show equal dimensions of these bones in normal subjects. In 42 patients with Turner's syndrome studied by Kosowicz,[8] "preponderance of the phalanges over the metacarpals," *i.e.*, a difference of 3 mm. or more, was noted in 57 per cent. This sign was positive in only 3 of 17 (17.7 per cent) observed cases of Klinefelter's syndrome, and unexpectedly in 60 per cent of our Japanese control subjects. Racial difference is a possible explanation of this high incidence of phalangeal preponderance in Japanese.

Fujita *et al.*[4] reported an incidence of brachymesophalangia with or without clinodactyly of the fifth finger in approximately 15 per cent of normal Japanese children, which is much higher than those reported among Caucasian children.[2,11] The somewhat low incidence seen in our Japanese controls might be due to age difference, our controls being of a higher age range.[4] In cases of Klinefelter's syndrome, 8 out of 17 observed cases (47 per cent) showed abnormalities of the fifth finger: 5 cases of brachymesophalangia with or without clinodactyly, and 3 cases of simple clinodactyly (Fig. 2; and 3). These figures appear significant, although they are somewhat lower

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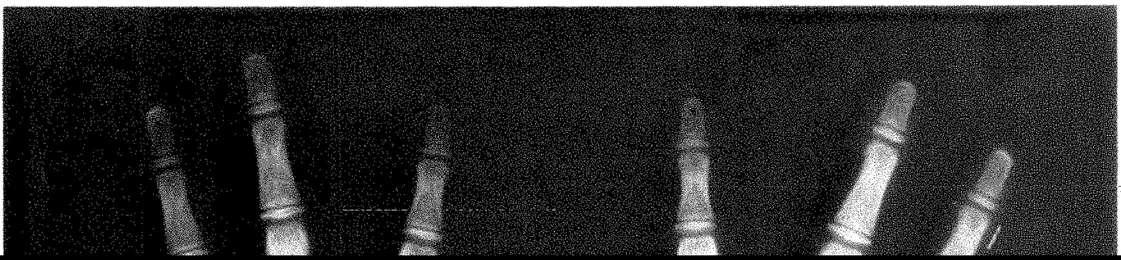

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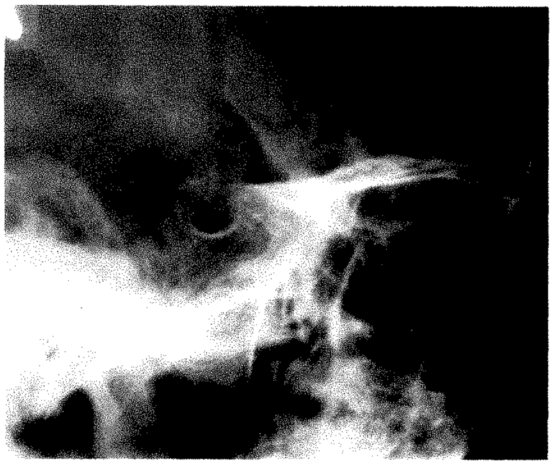

Fig. 4. Relatively small sella with overlapping of the prominent anterior and posterior clinoid processes in a 15 year old XXY male. The paranasal sinuses are poorly pneumatized.

with bilateral lumbar ribs, and 2 cases with very small or hypoplastic 12th ribs. There was no definite roentgenographic evidence of cardiovascular or pulmonary abnormalities on plain chest roentgenograms.

Seventeen cases had intravenous pyelography and none of these demonstrated significant abnormalities.

Spina bifida occulta of the first sacral segment was present in 7 of 16 observed cases.

Although no special view of the pelvic inlet was available, an attempt was made to determine the pelvis type from anteroposterior roentgenograms; 4 of 14 cases were estimated to be rather gynecoid in configuration.

Keats and Burns[6] mentioned that skeletal maturation in Klinefelter's syndrome proceeds normally while O'Hara[9] reported that retarded bone maturation is often found as a result of delay in epiphyseal closure.

In our series, estimation of the bone age was made in cases younger than 20 years according to the chart worked out by Sugiura and Nakazawa.[13] Up to 15 years of age, the roentgenograms of the hands and feet were used for evaluation, and the roentgenograms of the pelvis were added in the cases between 15 and 20 years of age. Four of 14 observed cases were thought to show retarded bone maturation. In 1 case of XXY, age 37 years, there was nonfusion of the proximal epiphysis of the clavicle bilaterally which should be fused with the shaft in the middle of the third decade (Fig. 5).

CONCLUSION

Klinefelter's syndrome is not infrequent and its diagnosis in pediatric or prepubertal age is extremely difficult.

The roentgenographic changes which have been described are mostly minor in nature, and usually not all evidence in any given case. Also, some of these changes such as the high metacarpal index and the delay of epiphyseal fusion, are probably

Table VI

SUMMARY OF ROENTGENOGRAPHIC FINDINGS IN KLINEFELTER'S SYNDROME

Roentgenographic Findings	XXY	XXXY XXXXY	XXYY	Total
Rib anomalies	2/13	2/2	1/2	5/17
Abnormal intravenous pyelogram	0/13	0/2	0/2	0/17
Spina bifida occulta (S-1)	5/13	1/2	1/1	7/16
Pelvic type				
Android	8/12	2/2	0/0	10/14
Gynecoid	4/12	0/2	0/0	4/14
Bone age				
Normal	7/10	1/2	2/2	10/14
Retarded	3/10	1/2	0/2	4/14

No. of positive findings/No. of observations.

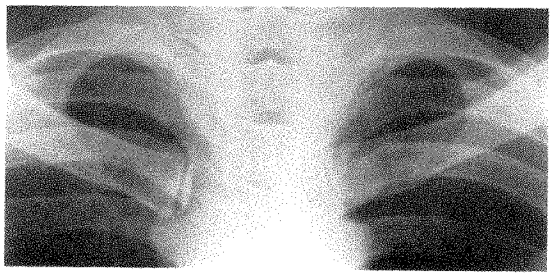

Fig. 5. A 37 year old patient with XXY Klinefelter's syndrome. Definite delay in fusion of the proximal epiphyses of the clavicle with the shaft.

secondary to hypogonadism, and not related to chromosomal aberration.

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LETHAL SHORT-LIMBED DWARFISM: ACHONDROGENESIS AND THANATOPHORIC DWARFISM*

By RONALD M. SALDINO, M.D.
SAN FRANCISCO, CALIFORNIA

ACHONDROGENESIS and thanatophoric dwarfism are congenital skeletal dysplasias characterized by short-limbed dwarfism. Both conditions are incompatible with life; all reported cases were either stillborn or died in the neonatal period. The infants are usually mistaken for examples of severe achondroplasia. The clinical features of achondrogenesis and thanatophoric dwarfism overlap and they cannot be clearly separated. The roentgenographic findings, however, are distinctive and diagnostic.

This study analyzes the anatomic and roentgenographic features in 2 siblings with achondrogenesis and compares them with an example of thanatophoric dwarfism. The available evidence in this family suggests that achondrogenesis is an autosomal recessive disease.

REPORT OF CASES

Case I. A 19 year old gypsy primagravida was admitted on March 11, 1959 in spontaneous labor, about 2 months before the estimated date of delivery. The prenatal course had been uneventful except for polyhydramnios. A stillborn female infant with short extremities was delivered.

The necropsy report described a 1,012 gram infant with the following measurements: length from crown to rump, 20 cm.; and from crown to heel 27 cm.; circumference of head, 30 cm.; of thorax 20 cm.; and of abdomen 25 cm.; length of arm from shoulder to fingertip, 9 cm.; and of leg from hip to ankle 7 cm. An epicanthal fold was present and the root of the nose was depressed. The neck was short and the abdomen prominent (Fig. 1*A*). Gross examination of the internal viscera revealed a patent foramen ovale and ductus arteriosus, nonaerated lungs, and an enlarged liver.

Histologic examination disclosed a prominent hematopoiesis in the liver, hypoplastic and atelectatic lungs, and abnormal bone morphology. The epiphyseal cartilage appeared lobulated and mushroomed, with increased cellularity and vascularity. Enchondral bone formation lacked an orderly pattern, and irregular capillary invasion and ossification were present. Formation of diaphyseal cortical bone appeared normal. Prominent cuffing of the epiphyseal cartilage resulted from prolongations of periosteal cortical bone. The histologic pattern of the vertebrae and ribs appeared similar, with only a small central area of ossification within the vertebral body (Fig. 3*A*). Little or no evidence of intervertebral disk formation was observed.

Case II. Ten years later the same gypsy again experienced spontaneous labor, 2 months before the expected date. A plain roentgenogram of the pelvis was obtained on May 19, 1969 because of polyhydramnios; a twin pregnancy was demonstrated. The roentgenographic appearance of one fetal skeleton was normal, whereas only the skull of the other fetus was visible. No exposure was known to teratogenic agents. Both fetal heart tones were heard. Artificial rupture of the membranes produced amniotic fluid of approximately 5,000 cc. The patient delivered a male, stillborn, short limbed-dwarf followed by a normal male infant of 3 pounds, 8 ounces. A tubal ligation was performed post partum.

At necropsy, the stillborn micromelic dwarf weighed 1,410 grams. Measurements were as follows: total length 28 cm.; length, head to coccyx 20 cm.; occipitofrontal circumference 31 cm.; thoracic circumference 22 cm.; abdominal circumference 25 cm.; right shoulder to fingertip 11.5 cm.; right hip to toe 12.1 cm.; heel to toe 5 cm.

Examination of the gross specimen revealed no external abnormalities other than a small right inguinal hernia and markedly shortened extremities (Fig. 1*B*). The hands and feet were short, but without other deformities. The peri-

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* From the Department of Radiology, University of California School of Medicine, San Francisco, California.
This investigation was supported by USPHS Grant AM 02589.

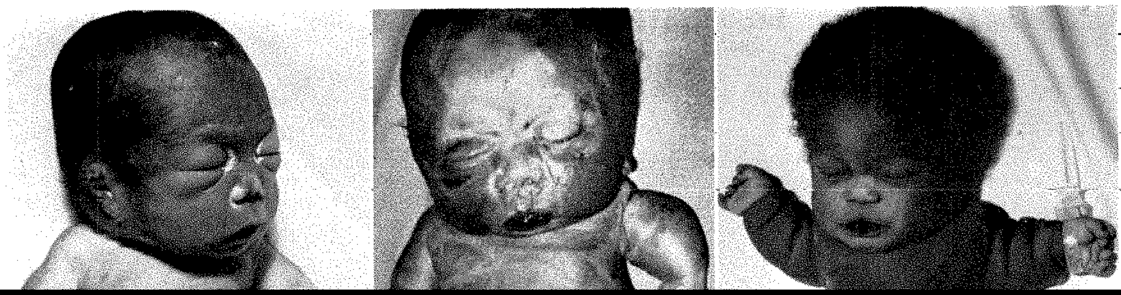

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Fig. 2. (*B*) The entire humerus, sagittally sectioned. Total actual length 3.5 cm. (*C*) Photomicrograph of chondro-osseous junction from another section of the humerus. Note lack of any columnar arrangement of cartilage cells, lack of matrix between expanded resting cartilage cells, and haphazard arrangement of primary trabeculae.

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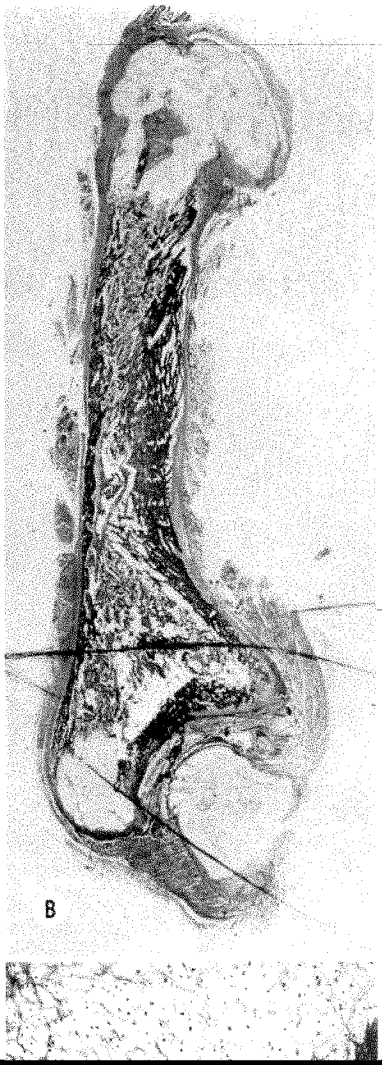
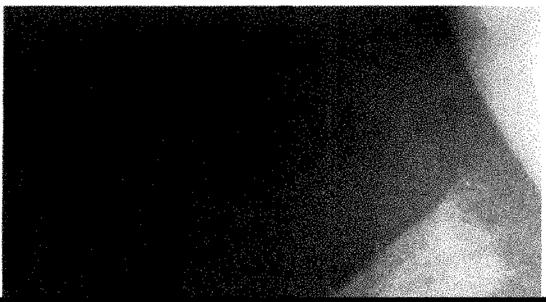


in relation to the relatively enlarged calvarium. In the frontal view, the soft tissues of the face were prominent and projected over the short neck. The scapulae were small and irregular, but the clavicles were normal. Shortening, slight flaring, and irregularity were seen in the anterior margins of the ribs. No sternal ossification centers were noted. Except for the sacrum, the neural arches throughout the vertebral column were faintly visualized. Roentgenographic ossification in the vertebral bodies



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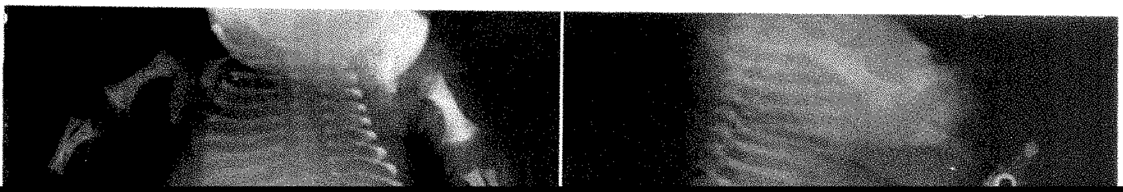

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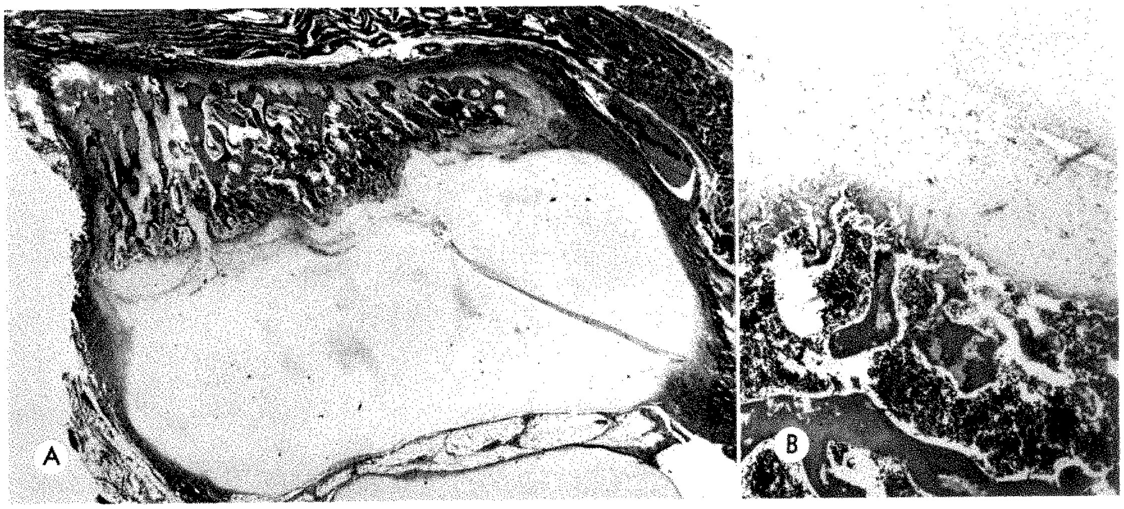

Fig. 8. Case iii. (*A*) Thanatophoric dwarfism. Section of distal femur. Lack of orderly enchondral ossification, greatly expanded epiphyseal cartilage, and absence of prolongation of the periosteal ossification as seen in achondrogenesis. (*B*) Photomicrograph of another section of the femur of thanatophoric dwarf

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described,[6,9,13,15,16,20,25] but achondrogenesis

appropriately received the designation of

[redacted]

Review of the sections of Cases I and II by Dr. D. Rimoin[32] led him to believe that the major abnormality was in the resting cartilage cells which were expanded and lacked surrounding matrix. In his opinion, the primary abnormality was not in enchondral ossification, but in the cartilage itself.

Occasionally, histologic sections in achondrogensis show a fibrous band of tissue interposed between the metaphyses and epiphyses. A similar band has been seen in thanatophoric dwarfism and in homozygous and heterozygous achondroplasia. It was also described in older reports of histologic material from stillborn dwarfs. The significance of this fibrous tissue is not clear. Probably it is related only secondarily to the primary process of retarded and disordered enchondral ossification. Recently 2 studies[15,16] of thanatophoric dwarfism with histopathologic material have been reported. Disorganization of enchondral ossification is seen in thanatophoric dwarfism[15,16] and homozygous achondroplasia.[10] The lack of cell matrix in the resting cartilage, however, seems to be specific for achondrogenesis. Not previously mentioned has been the lack of normal development of the intervertebral disk in achondrogenesis (Fig. 3, *A* and *B*).

Histologic examination of material from children with classic achondroplasia,[30,33] in contrast, does not show a disorganized pattern. Instead the arrangement is orderly, with an increased amount of matrix between the cartilage columns. Rimoin *et al.*[33] suggested that the basic defect in achondroplasia may be a quantitative decrease in the rate of enchondral ossification.

Achondrogenesis and thanatophoric dwarfism are both skeletal dysplasias that result in profound bony abnormalities in the neonatal infant. Several roentgenographic findings may be seen in both conditions. These include absent ossification of the talus and calcaneus, deficient or absent ossification of the sternum, short metacarpals, metatarsals, and phalanges, an enlarged cranium, and variable shortening of the ribs. The ribs of the thanatophoric dwarf are generally shorter and result in more narrowing of the thorax than is seen in achondrogenesis. Severe micromelia is present in both conditions. The shortening of the extremities in achondrogenesis may be so severe that length of the femur is only 3 cm. or less.

Although similarities exist between achondrogenesis and thanatophoric dwarfism, their roentgenographic differentiation is not difficult. The profoundly retarded skeletal ossification in achondrogenesis is unique and characteristic (Fig. 5, *A* and *B*; 6, *A* and *B*; and 7). In the normal fetus, roentgenographic evidence of ossification of the vertebral bodies, sacrum, ischial and pubic bones, calcaneus, and talus is present during the second trimester.[27,28] Because of lack of mineralization in achondrogenesis, these skeletal structures are not visible on the roentgenogram. Although the amount of ossification of the bones in achondrogenesis varies somewhat, the over-all roentgenographic pattern is diagnostic.

Distinctive roentgen features are demonstrated in the pelvic bones in achondrogenesis (Fig. 7; and 10, *A* and *B*). The sacrum and pubic bones are not visible, and the ischium also is either absent or only faintly seen. Height of the iliac bones is markedly decreased and the ilia lack normal flaring. The sacrosciatic notch is not narrow. Instead, a long, single arc curves along the medial aspect of the iliac bones ending in a short, sharp ischial spine. The acetabular roofs are flattened and the lateral margins of the ilia poorly defined.

The vertebral bodies in achondrogenesis are often completely absent or only barely visible on roentgenograms (Fig. 5, *A* and *B*; and 7). The vertebral bodies in the low thoracic and upper lumbar spine, the first to ossify in the normal infant, are usually the most clearly seen in achondrogenesis. The cartilaginous precursors of the sacrum and lumbar spine are present in achondrogenesis. In contrast, these precursors are missing in congenital absence of the sacrum or lumbar spine (caudal regression syndrome), which results in medial displacement and fusion of the ilia.[1]

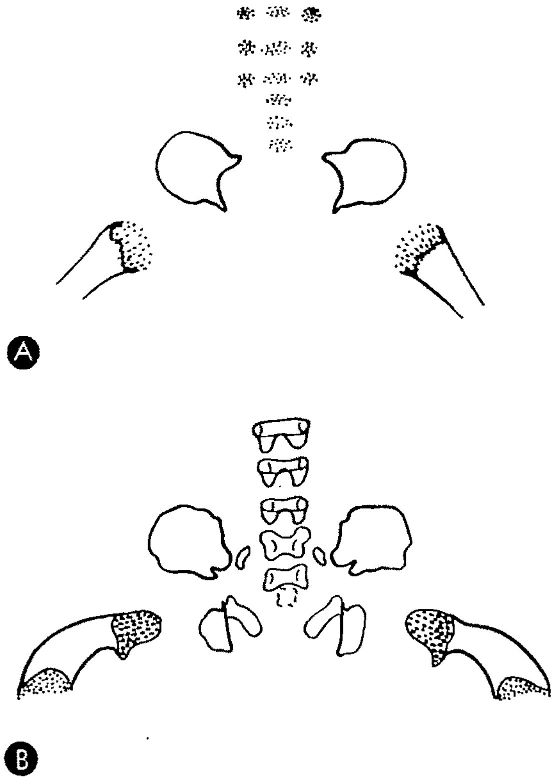

Fig. 10. Line drawings of the roentgenograms of the pelvis of (*A*) achondrogenesis and (*B*) thanatophoric dwarfism. In achondrogenesis (*A*) the vertebral bodies are barely perceptible. The iliac wings are small with sharply delineated long medial margins with no narrowing of the sacrosciatic notch. Ischial, pubic and sacral bones are absent. The metaphyseal margin is irregular and faint linear densities extend from the end of the bone, representing prolongation of periosteal ossification. In thanatophoric dwarfism (*B*) the vertebral bodies and sacrum are present, with marked flattening of the vertebral bodies. The iliac wings are small with a narrow sacrosciatic notch. The ischial and pubic bones are short and thick with spike-like projections of the ischia. Increased radiolucency of a triangular shape of the proximal femora is seen. Thorn-like bony spurs are present on the inner margin of the proximal femora.

The femora in achondrogenesis are shortened, but usually not bowed. The metaphyseal margins are irregular and not clearly outlined. Projections of periosteal ossification around the margins of the ends of the bones may appear as faint striations. The humeri are short with prominent, wide ends (Fig. 2*A*).

The roentgenographic features of thanatophoric dwarfism, the other type of lethal short-limbed dwarfism manifested at birth, are sufficiently distinct to permit differentiation from achondrogenesis (Fig. 9, *A* and *B*). In contrast to achondrogenesis, the thanatophoric dwarf has markedly bowed femora, a narrow sacrosciatic notch, and roentgenographically discernible vertebral bodies, sacrum, and pubic and ischial bones. Furthermore, the spine in thanatophoric dwarfism is distinctive. The intervertebral spaces are increased, the vertebral bodies severely flattened with constricted midportions, and the lumbar interpedicular distance lacks widening. Less widening of the metaphyseal ends is seen in the humeri in thanatophoric dwarfism.

Giedion[6] has stressed the horizontal thorn-like projections on the long bones, a constant finding in thanatophoric dwarfism. These bony spurs are most evident in the proximal femora, but are also seen in the ischia, proximal tibiae, and other long bones (Fig. 9*B*; and 10, *A* and *B*). An increased translucency of the proximal femora is similar to that in achondroplasia except that it is pear-shaped or triangular, whereas in achondroplasia it is oval. The appearance of the proximal femora in achondrogenesis and thanatophoric dwarfism differs greatly and serves to differentiate the diagnosis.

Bony "spurs" are present in both achondrogenesis and thanatophoric dwarfism. In achondrogenesis the spurs are prolongations of periosteal ossification at the margins of the metaphyses resulting in a cupped or concave appearance to the end of the bone (Fig. 4; 6, *A* and *B*; and 10, *A* and *B*). In thanatophoric dwarfism the spurs are bony, thorn-like projections in the region of the lesser trochanters, ischial bones, and proximal tibiae (Fig. 8, *A* and *B*; 9, *A* and *B*; and 10, *A* and *B*).

The differential diagnostic features of achondrogenesis and thanatophoric dwarfism are presented in Tables I and II. A diagrammatic representation of the pelvis and surrounding bones in these two condi-

Table I

CLINICAL AND ANATOMIC FEATURES OF LETHAL SHORT-LIMBED DWARFISM

	Achondrogenesis	Thanatophoric Dwarfism
History	Stillborn, often premature; frequent maternal polyhydramnios	Stillborn, or death in neonatal period; frequent maternal polyhydramnios
Clinical appearance	Short trunk, distended abdomen; extremities shortened, may be extreme; head size normal or enlarged	Normal trunk length, distended abdomen; extremities short and held in extended position; head size normal or enlarged
Total body length	Severe, over-all shortening (25 to 38 cm.)	Average 40 cm. (29 to 47 cm.); shortening especially of extremities
Genetics	Probably autosomal recessive; our Case II and case reported by Morii *et al.*,[26] each had a normal fraternal twin; one case associated with a teratogenic agent[14]	Unknown mode of inheritance; Case I reported by Keats *et al.*[16] had a normal fraternal twin
Familial examples	Present report only (Cases I and II)	None reported
Bone, histologic appearance	Disorganized enchondral ossification; abnormal resting cartilage cells; poor development of intervertebral disk; periosteal spurs at metaphyses	Disorganized enchondral ossification
Number of known cases	About 15	About 35

tions may be seen in Figure 10, *A* and *B*. Further discussion of the differential diagnosis may be found in the articles by Kaufman *et al.*,[15] Keats *et al.*,[16] Langer *et al.*,[19] Maroteaux *et al.*,[25] and others.[9,13]

Some delay in ossification in the sternum, pubis, talus, and calcaneus is a feature of spondyloepiphyseal dysplasia congenita. This disease, however, is compatible with life, and can be distinguished from achondrogenesis by other clinical and roentgenographic signs as described by Spranger and Langer.[35]

Of interest was the fact that our Case II of achondrogenesis and the one described by Morii *et al.*[26] each had a normal fraternal twin. Keats *et al.*[16] presented an example of thanatophoric dwarfism in one of fraternal twins, and two different mutations were present among siblings described by Huguenin *et al.*[13] Thanatophoric dwarfism has not yet been reported in siblings although a history of increased fetal abortions has been noted.

The present communication is the first that documents achondrogenesis in siblings. The 2 other "deformed stillborns" in the obstetrical history of the mother of Cases I and II also may have been examples of achondrogenesis, but this assumption has not been proved. The parents of these children were consanguineous in that the fathers of the parents were second cousins. No history of dwarfism in the family was known. The parents were young and no chromosomal abnormalities were detected in the mother. It is likely, therefore, that at least in this family, achondrogenesis was inherited through an autosomal recessive gene.

Table II

ROENTGENOGRAPHIC FEATURES OF LETHAL SHORT-LIMBED DWARFISM

	Achondrogenesis	Thanatophoric Dwarfism
Skull	Normal or enlarged with normal base	Normal or enlarged with mild frontal bossing and short base; narrow foramen magnum
Thorax	Short horizontal ribs; absent sternal ossification	Severely shortened, horizontal ribs with narrow thorax; sternal ossification may be delayed
Spine	Absent or severely retarded ossification of	Ossification of vertebral body present with

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THANATOPHORIC DWARFISM *IN UTERO**

A CASE REPORT

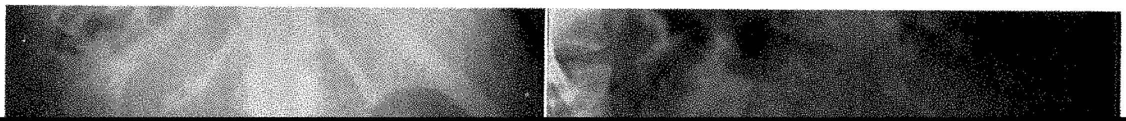
By ROBERT E. CAMPBELL, M.D.†
PHILADELPHIA, PENNSYLVANIA

UNTIL recently thanatophoric dwarfism, a short-limb dwarf condition, has been misinterpreted as a severe, immature form of achondroplastic dwarfism. In 1967, Maroteaux *et al.*[4] first stated that thanatophoric (death bearing) dwarfism is a definite, discrete entity. Two cases of thanatophoric dwarfism *in utero* diagnosed before delivery by roentgenographic examination have been recorded in the foreign medical literature[1,5] and recently another case was reported in the English language pediatric literature.[2] To my knowledge, thanatophoric dwarfism *in utero* has not been reported in the English language radiologic literature, and, therefore, this case is presented.

REPORT OF A CASE

B.B.W., a Negro male, was delivered at term by cesarean section on January 5, 1969. The pregnancy was normal until hydramnios developed during the seventh gestational month. Abdominal roentgenograms at that time revealed a small, abnormal fetus, "probably an achondroplastic dwarf."

The mother was 26 years old, the father 30. Both parents and their relatives were normal in stature. There was no history of consanguinity. The mother developed mild diabetes, controlled by an oral hypoglycemic agent, at the



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age of 23 years. The father was healthy. Two living siblings, 11 and 9 years old, were healthy

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EDITORIALS

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this training be at a Board-approved center?

The trainee must be made aware of the usefulness of the roentgen method in pediatric diagnosis as well as its, and his own, limitations in this field. He must be exposed to the special techniques required to handle infants and children, and must have experience in their use and in fluoroscopy. He must establish avenues of consultation that will allow him, in the knowledge of his own limitations, to obtain prompt and sympathetic consultation in dealing with more complicated pediatric problems. The training requirements, and facilities in which these training requirements are fulfilled, must again be carefully investigated and evaluated so that the needs of child and radiologist alike can best be served.

Bertram R. Girdany, M.D.
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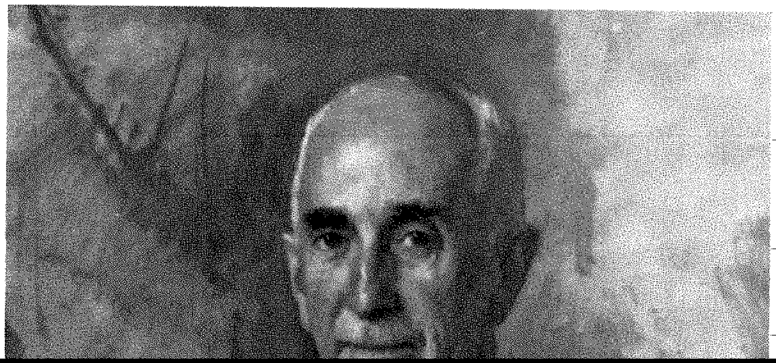

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the first book in English dealing exclusively with juvenile neoplasms.

He was a member of the American Medical Association, a former member of the New York Chapter, American Pediatrics Academy, a fellow in the New Jersey Chapter of the American Academy of Pediatrics, a member of the New York Academy of Medicine, the James Ewing Society, the American Radium Society, the Irish and American Pediatrics Society, and the New York Celtic Medical Society. Prior to his death he was Chairman Emeritus, Department of Pediatrics, Memorial Sloan-Kettering Cancer Center; Clinical Professor of Pediatrics Emeritus, Cornell University Medical College; and consulting pediatrician in 8 metropolitan hospitals. He took his medical postgraduate education at Bellevue Hospital in children's surgical services. Before his appointment to the staff at Memorial Hospital he was Attending Physician on the pediatrics staff of St. Lukes Hospital in New York.

Dr. Dargeon served in both the First and Second World Wars, retiring as Captain, Medical Corps, U. S. Naval Reserve. He participated in the Kwajalein, New Britain, Guam, Leyte, Moratsi, and Lingayen Gulf invasions and was awarded 6 Bronze Stars.

He is the recipient of several awards, including the 1969 Alumni Award of Albany Medical College, the James Ewing (Society) medal in 1968, and was the Henry Harrington Janeway medalist and lecturer of the American Radium Society in 1964. In 1961 he was the Otto A. Faust Lecturer, Albany Medical College. In 1935 he was elected to the American Board of Pediatrics and in 1950 to Theta Chapter, Alpha Omega Alpha.

He is survived by his wife and daughter, Jill.

M. LOIS MURPHY, M.D.

Memorial Hospital
444 East 68th Street
New York, New York 10021

NEWS ITEMS

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Professor Ueda, the newly elected president of the WFNMB, requested formally that the Society of Nuclear Medicine arrange a one-and-a-half day meeting of the WFNMB just before its own meeting June 26–July 2, 1971, in Los Angeles, California.

The Board of Trustees of the Society of Nuclear Medicine has agreed to do so. After a reception, a gala opening ceremony, and a banquet for the WFNMB will be held on Saturday evening, June 26, 1971. The WFNMB will hold its First Scientific Assembly on Sunday, June 27, 1971.

The scientific program will consist of contributed papers. Members of the Society of Nuclear Medicine are requested to submit abstracts of papers as soon as possible to Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, Washington University School of Medicine, 510 S. Kingshighway, St. Louis, Missouri 63110.

PHYSICAL ASPECTS OF NUCLEAR MEDICINE

This special course will be held July 26–30, 1971 at the Massachusetts Institute of Technology.

The course is designed to bring participants up to date on the state of the art in nuclear medicine, with emphasis on physical aspects. Topics to be covered include new developments in isotope techniques, instrumentation, data processing, clinical applications, specific organ studies, and therapy.

The course is sponsored jointly by M.I.T. and Harvard Medical School.

For further information please contact Director of the Summer Session, M.I.T. Room E19–356, Cambridge, Massachusetts 02139.

POSTGRADUATE COURSE IN DIAGNOSTIC ULTRASOUND

The Montreal General Hospital, a McGill University Teaching Hospital, will conduct a Postgraduate Course in Diagnostic Ultrasound on June 11th and June 12th, 1971.

This course will include lectures and the demonstration of equipment with emphasis on present knowledge and recent advances in the practical diagnostic use of Ultrasound. It will be of special interest to Radiologists, Cardiologists, Obstetricians, Neurologists and Neurosurgeons.

Please address inquiries to: The Secretary, Postgraduate Board, The Montreal General Hospital, 1650 Cedar Avenue, Montreal 109, Quebec, Canada.

BOOK REVIEW

Dictionnaire Français de Médecine et de Biologie. En quatre volumes. Volume I: A–D. By A. Manuila, L. Manuila, M. Nicole and H. Lambert: with the collaboration of J. Hureau (anatomie), J. Polonovski (chimie biologique), and 350 specialists. Preface by M. G. Candau, Directeur général de l'Organisation mondiale de la Santé. Publication

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lications of WHO, and his distinguished co-workers have spent 14 years in preparing this epoch-making contribution to the French language medical literature.

There are several innovations in this dictionary which are unique. Some of these are: (1) it encompasses a collection of dictionaries of various specialties, thus serving as a veritable med-

BOOKS RECEIVED

Bone Marrow Transplantation and Leucocyte Transfusions. By Georges Mathé, Professor of Experimental Oncology and Director, Institute of Cancerology and Immunogenetics, Hôpital Paul-Brousse, Villejuif, France; Jean-Louis Amiel, Professor of Oncology, Hôpital Paul-Brousse; and Leon Schwarzenberg, Hemobiologist at the Hôpital Paul-Brousse. American Lecture Series No. 793. Cloth. Pp. 206, with some figures. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

Radiological Physics Examination Review Book. Volume I. Edited by Colin G. Orton, Ph.D., Assistant Professor of Radiology, and Senior Physicist; and Christopher H. Marshall, Ph.D., Assistant Professor of Radiology, and Radiation Safety

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Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

Production des Rayons X; Leur Application en Radiographie Médicale et Industrielle. By Yves Duriez, Ingénieur I.E.G. Paper. Pp. 158, with some figures. Price, 18 F. Masson et Cie, Éditeurs, 120 Boulevard Saint-Germain, Paris, France, 1971.

Angiography of the Spinal Cord. By R. Djindjian, with the collaboration of M. Hurth, R. Houdart, G. Laborit, H. Julian and H. Mamo. English Translation by Irvin I. Kricheff, New York. In French and English. Cloth. Pp. 482, with many illustrations. Price, $42.50. University Park Press, 115 Chamber of Commerce Building, Baltimore, Md. 21202, 1970.

Technique of Lymphography and Principles of

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SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Sheraton Hotel, Boston, Mass., September 28–October 1, 1971.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Boca Raton Hotel and Club, Boca Raton, Fla., May 14–19, 1972. Conjoint meeting with the James Ewing Society and the American Society for Head and Neck Surgery.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 28–December 3, 1971.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: St. Louis, Mo., Chase-Park Hotel, March 30–April 3, 1971.

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pital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

SECOND CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY.
President, Professor Dr. J. R. von Ronnen, State University of Leiden, The Netherlands.
Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, The Netherlands. Congress Meeting: Amsterdam, The Netherlands, June 14–18, 1971.

FIRST ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia. Meeting: Melbourne, Australia, Nov. 22–26, 1971.

ALABAMA CHAPTER OF ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

75th St., Apt. 11-A, New York, N.Y. 10021. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

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Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130

Memphis Roentgen Society

Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society

Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 p.m. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR

Secretary, Dr. David P. Corbett, Harper Hospital, Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 p.m.

Mid-Hudson Radiological Society

Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 p.m., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society

Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 p.m.

Missouri Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR

Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101.

Nebraska Chapter of ACR

Secretary-Treasurer, Dr. N. Patrick Kenney, 3618 S. 114th Ave., Omaha, Nebr. 68144. Meets third Wednesday of each month at 6 p.m. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR

Secretary, Dr. Harris W. Knudson, 2020 W. Charleston Blvd., Las Vegas, Nev. 89102.

New England Roentgen Ray Society

Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 p.m.

New Hampshire Roentgen Ray Society, Chapter of ACR

Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR

Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society

Secretary-Treasurer, Dr. Samuel H. Madell, 1. E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 p.m. Annual Spring Conference: Waldorf Astoria Hotel, New York, April 29–May 1, 1971. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR

Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

North Carolina Chapter of ACR.

Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR

Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society

Secretary, Dr. John W. Morris, III., Department of Radiology, Halifax District Hospital, Daytona Beach, Fla. 32015. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society

Secretary, Dr. Barbara Chick, Glens Falls Hospital, Glens Falls, N.Y. 12801. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiological Society

Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern Ohio Radiological Society

Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR

Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

Oklahoma State Radiological Society, Chapter of ACR

Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society

Secretary, Dr. Edward I. Miller, 301 Newport Blvd., Newport Beach, Calif. 92660. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Clinton B. Sayles, 214 Medical Dental Bldg., Portland, Ore. 97205. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society

Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society

Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

Pennsylvania Radiological Society, Chapter of ACR

Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society

Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 p.m., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society

Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society

Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting: Boston, Mass., May 9–13, 1971.

Radiological Society of Connecticut, Inc., Chapter of ACR

Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly

Radiological Society of Greater Cincinnati

Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City

Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07206. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 19–21, 1971.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, 20 Bridge Rd., Kentfield, Calif. 94904. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Raymond L. Schwinn, 480 Monterey Ave., Los Gatos, Calif. 95030. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Sheraton Hotel, Boston, Mass., September 26–27, 1971.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Haakon O. Haugan, 716 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Sixteenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 28–30, 1972.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Lawrence R. Nickell, Maury County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Flagship Hotel on Pier, Galveston, Tex.

The Fleischner Society
Secretary-Treasurer, Eric N. C. Milne, M.B., Medical Sciences Bldg., University of Toronto, Ontario, Canada. Meets in Williamsburg, Va., at the Williamsburg Conference Center, in conjunction with a course on "Modern Trends in Roentgenology of the Chest," sponsored by the Virginia Commonwealth University, Richmond, Va., March 14–18, 1971.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. R. Newell Ford, St. Mark Hospital, 803 North 2nd West, Salt Lake City, Utah 84103. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR

Secretary, Dr. Edward A. Kupic, Mary Fletcher Hosp., Burlington, Vt. 05401.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Joan Wohlgemuth, 5021 Seminary Rd., Alexandria, Va. 22311.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Leland L. Burnett, 5765 S. Eddy St., Seattle, Wash. 98118. Meets quarterly.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Octavio Toussaint Goribar. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 p.m. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting, Oxford, England, July 2–3, 1971.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. F. Robert MacDonald, *Associate Honorary Secretary*, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting: Palliser Hotel, Calgary, Alberta, March 15–19, 1971.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Víctor A. Añaños, Instituto de Radiología, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 p.m. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología
Secretary-General, Dr. Juan R. Heilbuth Pacheco, Santa Fe 1171, Buenos Aires. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoêdo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Ricardo Cortez, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raúl Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología

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Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each

ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

Head

Oldham, Robert K., and Staab, Edward V. Aseptic meningitis following the intrathecal injection of radioiodinated serum albumin. *Radiology*, Nov., 1970, *97*, 317–321. (From: Departments of Radiology and Medicine, Vanderbilt University Hospital, Nashville, Tenn.)

Two cases of aseptic meningitis following RISA cisternography are reported. Less than 2 mg. of albumin iodinated with 100 microcuries I^{131} (high specific activity RISA) was injected in each case.

Both cases developed fever and stiff neck within 24 hours and spinal fluid showed moderate pleocytosis, predominantly polymorphonuclear leukocytes in one case and all mononuclear cells in the other. Cerebrospinal fluid protein elevations were 254 mg. per cent and 83 mg. per cent.

The symptoms and signs in both cases subsided rapidly and no permanent sequelae have been noted.

In Case 1, pleocytosis occurred after the first RISA cisternography and was more pronounced following the second examination 3 months later, suggesting an anamnestic response.

These 2 cases represent an incidence of 3 per cent of the authors' series. Isolated case reports of similar cases have appeared in the literature.—*William H. Roush, M.D.*

Taddei, L., Cionini, L., and Brancato, R. Oculo-palpebral lesions following high-speed electron treatment of maxillary sinus tumours. *Clin. Radiol.*, Oct., 1970, *21*, 417–424. (From: Departments of Radiology and Ophthalmology, University of Florence, Italy.)

The authors report 3 patients who were treated for tumors of the maxillary sinus with high-speed electrons from a 3 mev. betatron including the orbit in the irradiated volume.

In these patients, who were normal prior to therapy, severe chronic inflammatory changes localized mostly in the anterior portion of the bulb developed. The lesions were severe enough to result in loss of vision and pain which made enucleation of the irradiated eye necessary.

Irradiation of the same volume with telecobalt beam using a wedge filter technique in 19 patients kept under observation for a period of 1 year produced no significant changes.

It is concluded that whenever it is considered necessary to include the orbit in the volume to be irradiated, the use of telecobalt with a wedge filter technique is preferable to the use of high speed electrons.—*S. A. Kaufman, M.D.*

Neck and Chest

Wright, F. W. Accidental injection of dionosil into the neck during bronchography. *Clin. Radiol.*, Oct., 1970, *21*, 384–389. (From: Churchill Hospital, Oxford, England.)

The unpleasantness of translingual catheterization is avoided by using the cricothyroid route for bronchography. Complications of this technique include surgical emphysema and injection of contrast medium into the soft tissues of the neck. The latter complication is uncommon, but 10 cases which have been collected over a considerable period of time are presented as a guide to management. Other cases from previous reports are summarized.

There were varying degrees of morbidity described including dysphagia, muscle spasm, and stiffness of the neck, pain, local tenderness, and hoarseness. Tracheostomy was necessary on a patient who had a tracheal tumor and 1 patient developed stridor.

In the discussion, the author indicates that this complication should never occur if the basic elementary rule of making certain that the needle tip at all times lies within the trachea before injection is observed. Aspiration of air after every 1 to 2 ml. of injection is the single best criterion to use, and in each complication described by the author this air withdrawal test was not properly carried out. He does not recommend the procedure in patients under 12 years of age.

Treatment of this complication includes hospital admission, sedation, and analgesia, with the attending personnel being alert for the possibility of supervening airway obstruction. Most of the symptoms of dysphagia and stiffness of the neck improve within a few days and the dionosil is usually completely absorbed in 2 to 8 weeks.—*Robert P. Andrews, M.D.*

Murphy, Raymond L. H., Barber, Doris, Broadhurst, Alice, and Bird, Kenneth T. Microwave transmission of chest roentgenograms. *Am. Rev. Resp. Dis.*, Nov., 1970, *102*, 771–777. (From: Department of Medicine, Massachusetts General Hospital, Boston, Mass.; and Middlesex County Hospital, Waltham, Mass.)

Utilizing a telemedical technique which consisted of a 2 way closed circuit television with transmission of the signal by a microwave, comparisons were made of the interpretations of roentgenograms viewed directly and after transmission by the closed circuit system. A remotely controlled plubicon camera with zoom lens focus control was used to obtain panoramic and detailed views of each roentgenogram.

Three observers viewed the roentgenograms by the Telediagnosis system assessing each roentgenogram independently. After discussion, a panel interpretation was agreed upon and recorded. The panelists were asked to describe the roentgenograms with regard to the presence and extent of abnormalities consistent with tuberculosis according to standard criteria. To assess intraobserver variability, the hospital radiologist reread the roentgenograms both directly and by the Telediagnosis system. There was no disagreement between the direct interpretation by the radiologist and those made by television in 90 per cent of the cases. Minimal disease was not overlooked and the principal difference in interpretation was in minor variations between minimal and moderately advanced, or moderately advanced and far advanced disease. Comparing the observations of the panelists and the radiologist, there was no disagreement between the classifications made in 77 per cent of the cases.

The major difference was the tendency to classify disease one category greater in severity than was recorded on the hospital chart. This tendency to "over read" the roentgenograms by television was greater than "under reading."

The capability of the zoom lens was shown to be useful in providing increased magnification.

The control of contrast and brightness on the television monitor allowed accentuation and diminution of features on the roentgenograms not easily attainable by conventional roentgenographic methods.

Observer variability was the principal difficulty encountered in this study which made statistical evaluation difficult and it was felt that this variability could mask variation due to the Telediagnosis system.

A review of the errors suggested that if this variation did occur it was small.—*James R. Knapp, M.D.*

Morrison, W. J., Wetherill, S., and Zyroff, J. The acute pulmonary edema of heroin intoxication. *Radiology*, Nov., 1970, *97*, 347–351. (From: The Johns Hopkins School of Medicine, Baltimore, Md.)

Heroin addiction in the United States poses an ever increasing problem manifested by both the growing number of addicts and the increasing social and political concern.

The acute intoxication phenomenon, frequently accompanied by characteristic pulmonary edema, is being recognized with increasing frequency as one of the major complications of heroin usage.

Thirty-seven cases of heroin-related disease were admitted to the Johns Hopkins Hospital between January 1964 and April 1969, with 2 of the users having required 2 separate hospitalizations. These 37 patients do not account for the entire hospital experience. Thirty-one per cent were male and 6 were female; 29 were Negro while 8 were Caucasian.

Among these heroin users, 10 suffered the acute intoxication syndrome, and in 8 of these severe, but rapidly clearing pulmonary edema developed.

The roentgenographic appearance of a bilateral edema pattern is illustrated. Previous reports have emphasized the occurrence of this pattern, sometimes with superimposed pneumonic infiltrates accompanying an acute heroin overdose. The edematous reaction in the lungs is rapid in onset and most commonly very severe.

The etiology of the pulmonary edema remains uncertain, but the possibility of a hypersensitivity reaction seems reasonable.—*Gung Eung Kim, M.D.*

Wesenberg, Richard L., Graven, Stanley N., and McCabe, Edward B. Radiological findings in wet-lung disease. *Radiology*, Jan., 1971, *98*, 69–74. (From: Department of Radiology, St. Mary's Hospital Medical Center and the Department of Pediatrics, University of Wisconsin Medical Center, Madison, Wis.)

The lung of a normal term fetus contains approximately 80 to 110 ml. of fluid. In the course of a normal vaginal delivery, up to one-third of this fluid is expressed from the lungs and out of the trachea by thoracic compression during passage through the birth canal. The remainder of the normal lung fluid is rapidly absorbed into the perivascular lymphatics and the pulmonary capillaries. The high incidence of respiratory distress in infants delivered by cesarean section might be due in part to lack of normal thoracic squeeze occurring during vaginal delivery.

The authors reviewed a series of 20 infants, 12 of whom were delivered by cesarean section, and give the details of 2 cases. All of these had clinical and roentgenographic findings consistent with delayed resorption of lung fluid—a condition the authors call wet-lung disease.

In the series, there was an equal sex distribution with gestational ages ranging from 32 to 40 weeks, with a mean age of 35 weeks. The primary indication for cesarean section was ante partum bleeding.

The clinical pattern observed most often was one in which the respiratory rate was normal in the first hour of life and gradually increased during the next 4 to 6 hours. The respiratory rate returned to normal in from 12 to 72 hours. None of the infants had heart murmurs or clinical signs of congestive heart failure. A new finding noted was hypoproteinemia in 9 patients in whom serum proteins were determined. There were no elevated hematocrits in the series. Eighteen of the 20 infants survived with supportive care.

The roentgenographic pattern of wet-lung disease, as demonstrated by serial roentgenograms in this series of cases, appeared to be quite consistent. The earliest findings (on roentgenograms taken at 2 to 6 hours of age) were a pattern of diffuse bilateral alveolar edema with concomitant hyperaeration of the lungs, sternal retraction, and an air bronchogram effect. This is followed by a transitory stage of clearing of the alveoli, with the development of a bilateral granular or miliary pattern. This is suggestive of hyaline membrane disease, but the lung fields gen-

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days after embolism and more slowly during the next 2 to 3 weeks. Failure to improve during the first month after embolism may be due to associated chronic disease but also to recurrent embolism or of the patients and 28 per cent demonstrated complications. There were contraction secondary to fibrosis, cystic disease, atelectasis, pneumothorax, pleural effusion, cavitation, and mycetoma formation. There was an increased frequency of complications among the Negroes. Contraction secondary to fibrosis was found in 20 per cent and bullous and bronchiectatic air cysts were found in approximately one-sixth of the patients. Cavitation can occur with a pyogenic infection or tuberculosis in previously compromised lung parenchyma, and further necrosis and cavity formation can occur. Fibrotic areas may undergo necrosis and then cavitate, and infection may cause thickening of emphysematous bulla walls and then this acts as, or actually becomes, a cavity. Mycetomata were discovered in 10 of the 25 cavitative cases and 8 of these patients had been treated at some time with steroids. The usual organism was *Aspergillus*. There seems to be a consistent relationship between hemoptysis and mycetoma formation. Atelectasis was an infrequent finding and an endobronchial component of the disease was probably necessary for it to occur.

The authors conclude that sarcoidosis patients with lymphadenopathy should be closely followed in anticipation of pulmonary parenchymal disease. The appearance of fibrosis and cyst formation may signal a change from a benign to a possibly debilitating chronic pulmonary disease. Routine tomography is suggested in patients with fibrosis to rule out the serious complication of mycetoma formation.—*Robert P. Andrews, M.D.*

GRAINGER, R. G., CASTELLINO, R. A., LEWIN, K., and STEINER, R. N. Hytrast: experimental bronchography comparing two different formulations. *Clin. Radiol.*, Oct., 1970, *21*, 390–395. (Address: R. G. Grainger, Department of Radiology, Royal Hospital, Sheffield 1, England.)

The authors found that there are 2 different hytrast formulations available, one of which is marketed in North America and another which is marketed in France and South Africa.

The American product is considerably less viscous, and it was thought that this might be responsible for a reported increased incidence of pneumonia after use of the American manufactured hytrast.

The authors decided to perform a study of bronchography in dogs using French hytrast, American low-viscosity hytrast, and dionosil oily. Segmental bronchography was carried out in 22 dogs. They found 38 lungs which they considered acceptable for the study and the findings in these lungs formed the basis of the report. The roentgenographic quality of the bronchograms was good in all cases, although there was an increase in the degree of alveolarization with hytrast of low viscosity (American) and oily dionosil. The quality of the study was considered better with both types of hytrast as compared to the dionosil. Microscopic changes consisted of pulmonary collapse and pneumonia. The amount of consolidation present was related to the period of post bronchographic survival. Microscopically there was bronchiolitis and bronchopneumonia in the animals sacrificed in the first 4 days. After this interval the presence of a predominantly chronic inflammatory reaction was noted, and in a few animals there was a mild interstitial fibrosis.

Histologically the bronchographic media are seen as birefringent brown crystals and these crystals were found in the interstitial tissues of the animals sacrificed after 4 days. Some crystals were found also in the upper lobes, although these areas of the lung were deliberately not injected with contrast medium, in order to have them serve as controls for the study.

The authors could find no significant difference in the pathologic response elicited by the 3 contrast media, although this does not agree with other reports. In their discussion, the authors point out that using the more viscous hytrast produced a degree of improved roentgenographic contrast, but that if one lowered the viscosity by dilution, increased alveolar flooding occurred. Alveolarization with hytrast is undesirable because the underlying chemicals cannot be hydrolized in the lung and tend to remain in crystal form in the alveoli and small bronchioles, so that a foreign body reaction can ensue. The dionosil can be hydrolized and the amount which is not expectorated is excreted.

The authors believe that the less viscous American manufactured hytrast produces a more rapid bronchogram but at the expense of alveolarization, and there is a consequent problem of hytrast crystal retention in the alveoli. This hypothesis was not confirmed by the animal experiments however; and they felt that this discrepancy may be due to the limitations of pathology and radiology in detecting minor changes in response, and also to the small number of animals used. They did note the following differences: (1) Three times more sodium carboxymethylcellulose in the French product as compared to the American hytrast; (2) the French product is twice as viscous; and (3) the degree of alveolarization is greater with the American product because of its decreased viscosity.

It is concluded that the clinical observations on the American hytrast will not necessarily apply to the original French product due to their underlying differences. Alveolarization is undesirable because of the lung's inability to effectively handle the hytrast in the alveoli, and this should be avoided. The French hytrast is of superior radiographic quality.—*Robert P. Andrews, M.D.*

GREYSON, N. DAVID, and ROSENTHALL, LEONARD. Detection of postoperative bronchopleural fistulas by radionuclide fog inhalation. *Canad. M. A. J.*, Dec., 1970, *103*, 1366–1368. (Address: Leonard Rosenthall, M.D., Division of Nuclear Medicine, Montreal General Hospital, Montreal 109, Quebec, Canada.)

Bronchopleural fistulas occur most frequently as a

Laboratory, University of California, Berkeley, Calif.)

Cesium 129 is produced by irrad~~iating sodium~~ iodide (NaI) with alpha particles in the cyclotron. The nuclear reaction is a (α, 2n) conversion of iodine 127 to cesium 129. The thresh~~old energy is 14.8~~

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Pulmonary artery banding is performed in infants and children with left-to-right shunts and pulmonary hypertension as a palliative procedure, with definitive surgery and removal of the band done at a later more optimal time.

This study includes 15 children who did not have preoperative selective right ventriculography, but who had cardiac catheterization and angiocardiography 1 to 6 years after pulmonary artery banding; 5 of these were again studied following removal of the band and complete surgical correction of the ventricular septal defect 1 to 2 years previously.

Normal valve leaflets were observed angiographically only in those 2 patients in whom the band was 15 mm. or more from the pulmonary valve or in the single patient in whom the band constriction was minimal. Abnormalities included: (a) distinct thickening of one or all leaflets of the pulmonary valve; (b) thickening of the free edge of the valve leaflets with distinct nodularity of its free margins; (c) pseudodoming of the pulmonary valve; and (d) poststenotic dilatation of the pulmonary artery.

The authors speculate that the band which is too tight or too close to the pulmonary valve interferes with normal valve motion which may eventually result in the above changes persisting after removal of the band and hence causing a residual pressure gradient. They suggest that, if possible, the band should be applied as far distal as possible on the pulmonary artery.—*L. M. Nikolai, M.D.*

Lavaurs, G., and Riitano, F. Les accidents de la coronarographie sélective. (Complications of selective coronary angiography.) *Ann. de radiol.*, 1970, *13*, 667–670. (From: Service de Radiologie du Centre Cantini, 60, avenue Roland-Garros, 13-Marseille-09, France.)

Complications of selective coronary arteriography tend to decrease with experience, but it is important for the entire diagnostic team to be prepared to deal rapidly with such events.

A review of 150 personal cases included 4 cardiac arrests after fibrillation, of which 2 were quickly reversed by external massage. Three of the arrests followed right coronary injection; in all 4 there was highly intense opacification of the sinus node. In 1 fatal case, a catheter had been placed far along the left coronary artery; the other patient suffered from far advanced myocardial disease.

Careful catheter positioning, test injection, and decreased injection pressure are now used to avoid such accidents.

Rapid reversal of ventricular fibrillation by defibrillator shock in 3 cases underscored having this machine ready for immediate use.

The rare neurologic complications are usually secondary to cardiac accidents.

Infection, hemorrhage, and thrombosis are hazards of the act of catheterization.

The 5 instances of loss of radial pulse (2 leading to surgery) were all encountered in the first 30 studies.

Finally, possible intolerance of the iodine contrast medium is recalled.—*Alan E. Oestreich, M.D.*

ABDOMEN

Keller, Rhona J., Wolf, Bernard S., and Khilnani, Mansho T. Roentgen features of healing and healed benign gastric ulcers. *Radiology*, Nov., 1970, *97*, 353–359. (From: Department of Radiology, The Mount Sinai Hospital and Mount Sinai School of Medicine, New York, N. Y.)

About 80 per cent of acute gastric ulcers are usually superficial in nature and heal rapidly within 2 to 4 weeks, leaving little or no trace pathologically. These ulcers are rarely demonstrable roentgenographically. Of the remainder, a number enter the chronic phase, and, because they have penetrated through the muscularis mucosae and submucosa and in most cases have extended into the muscularis propria, are usually demonstrable roentgenographically. Healing of these ulcers is due to contraction of the muscular fibers as well as bridging by and subsequent contraction of fibrous and elastic tissues, leaving a relatively small surface area covered by regenerative epithelium which is less complex in character and thinner than the normal gastric mucosa.

The size of this residual scar is directly related to the size of the original ulcer crater with small ulcers leaving only a small dimple while larger ulcers will heal leaving a relatively larger flat segment of limited distensibility. During the healing process, and often for as long as 2 and 3 years following healing of the ulcer, and particularly of ulcers on or near the greater curvature, a pattern of smooth sharply defined, radiating folds, extending in tapering fashion to the margin of the healing ulcer or scar, is noted. Healing ulcers on or near the lesser curvature usually do not induce the production of a well defined radiating fold pattern.

The pattern of healing of a benign gastric ulcer may be complicated by at least 3 factors: the unusual size of the original ulcer crater; the portion of the stomach in which it arises; and the chronicity of the process. The segment of the gastric wall where the residual scar is formed, and which represents the re-epithelialized crater, is less pliable, but this may completely disappear after 3 or 4 years. Very large or chronic ulcers may give somewhat unusual appearances due to interrupted and uneven closure of the mucosal defect simulating superficial carcinoma.

The differential diagnosis of this residual gastric wall deformity should include superficial carcinoma of the stomach, and either persistent active, or recurrent ulcer disease. The ulcer crater in a carcinoma of the stomach, if roentgenographically apparent, is often irregular and usually ill-defined. If a radiating

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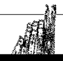

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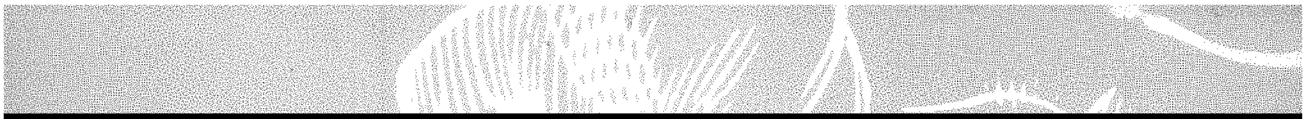

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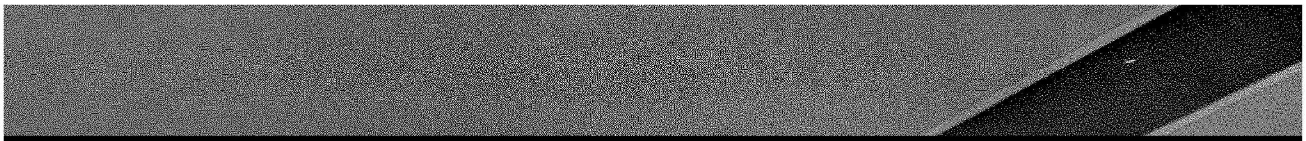

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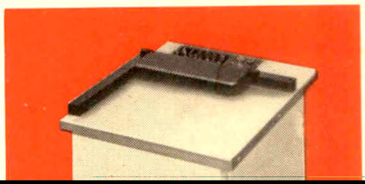

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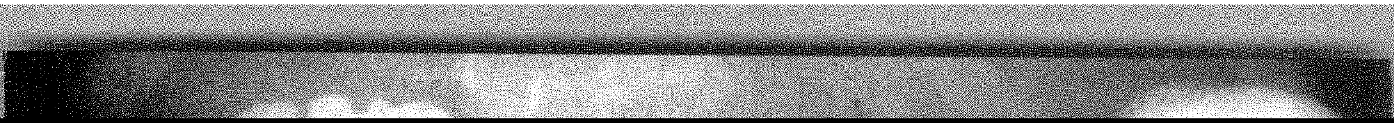

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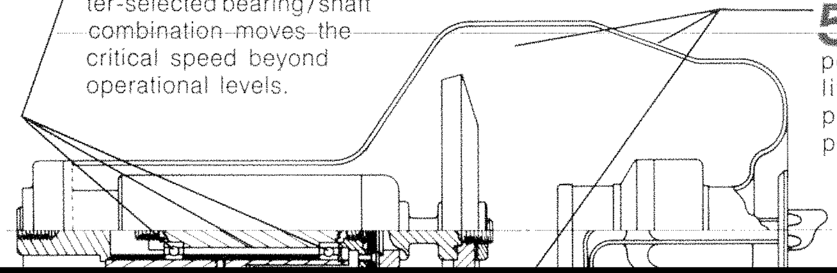

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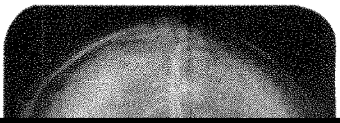

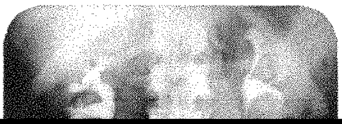

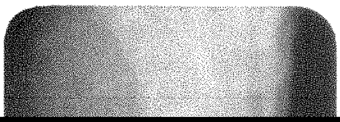

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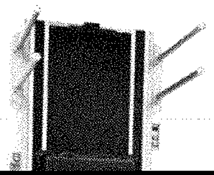

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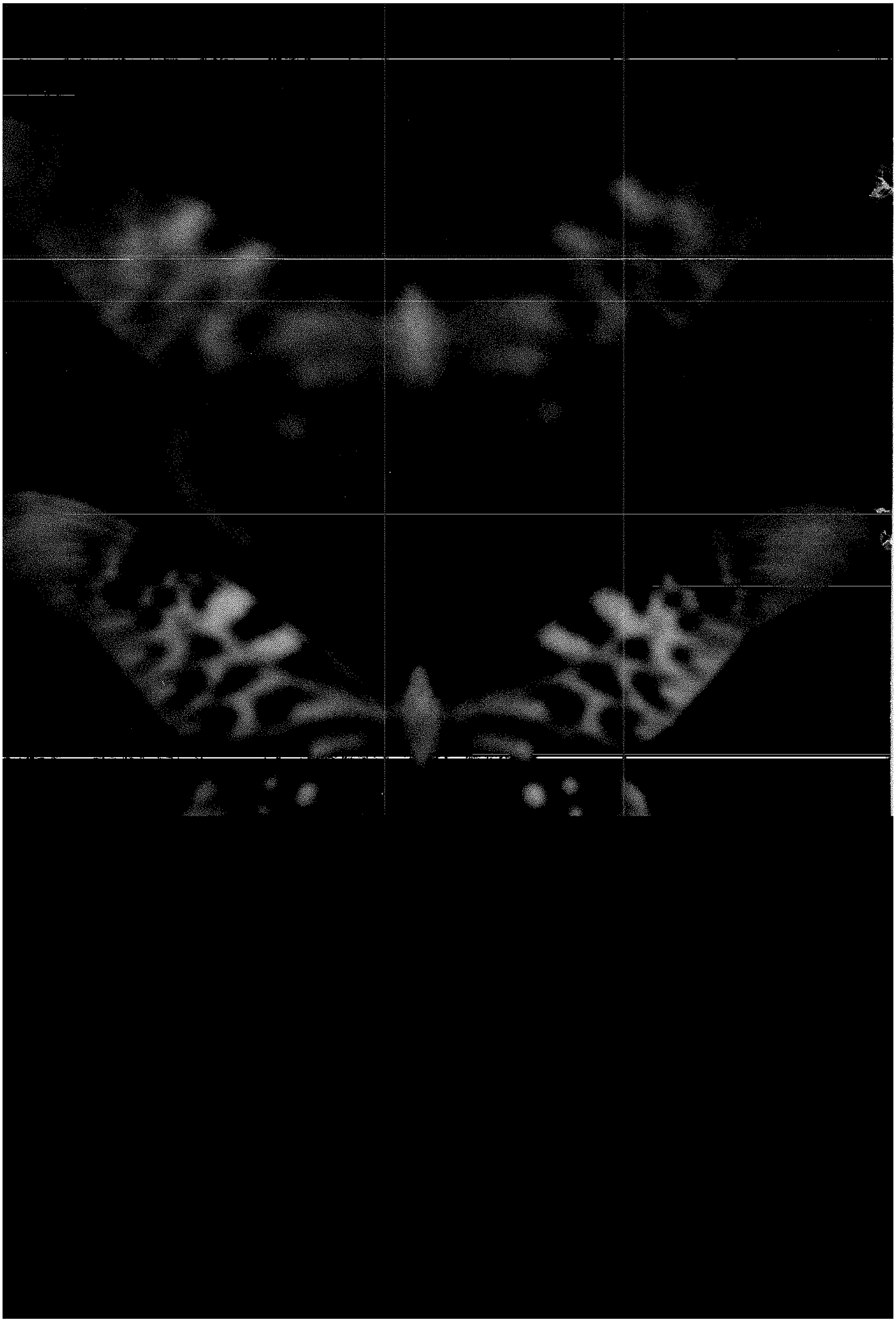

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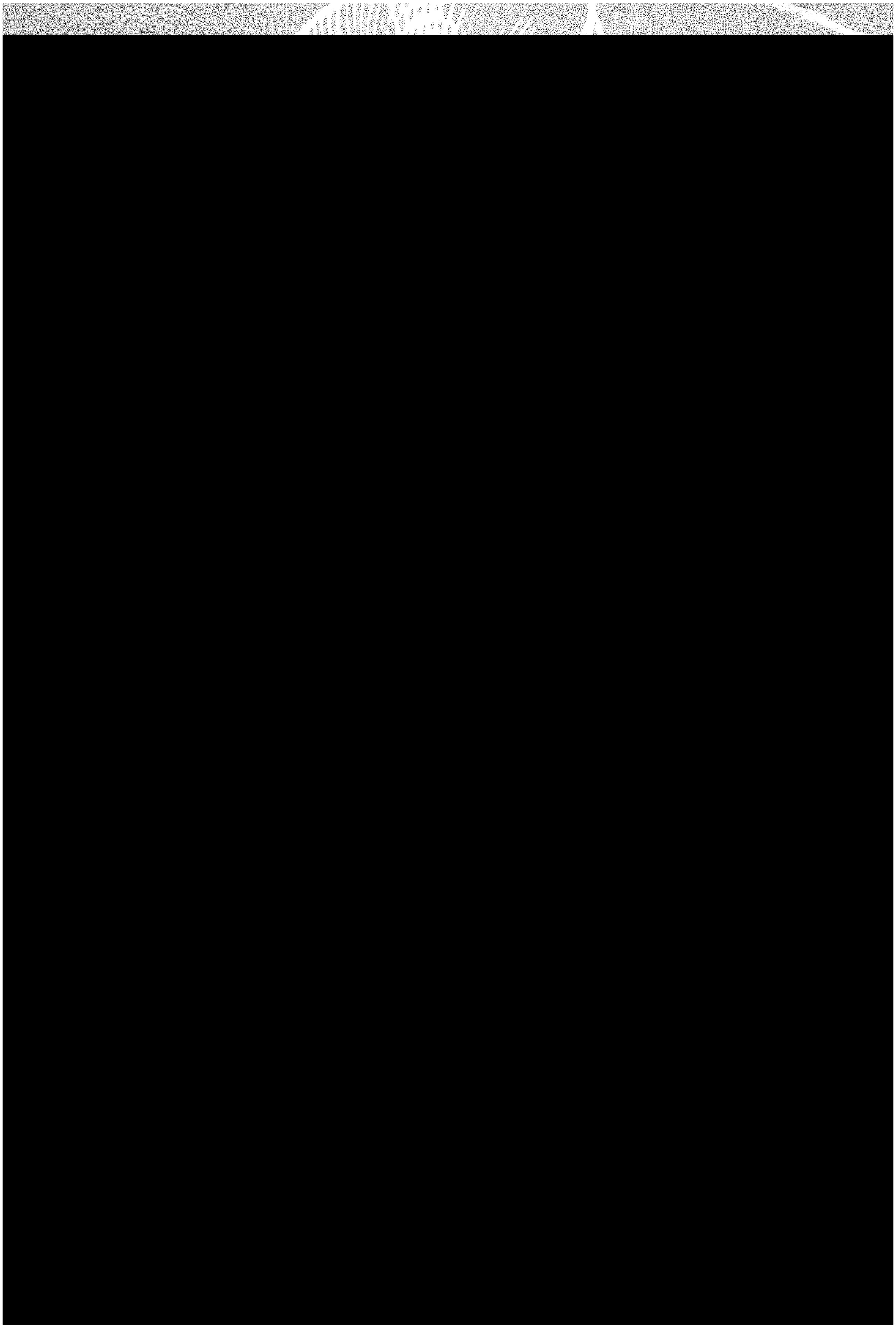

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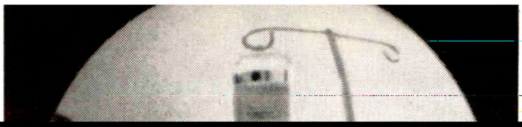

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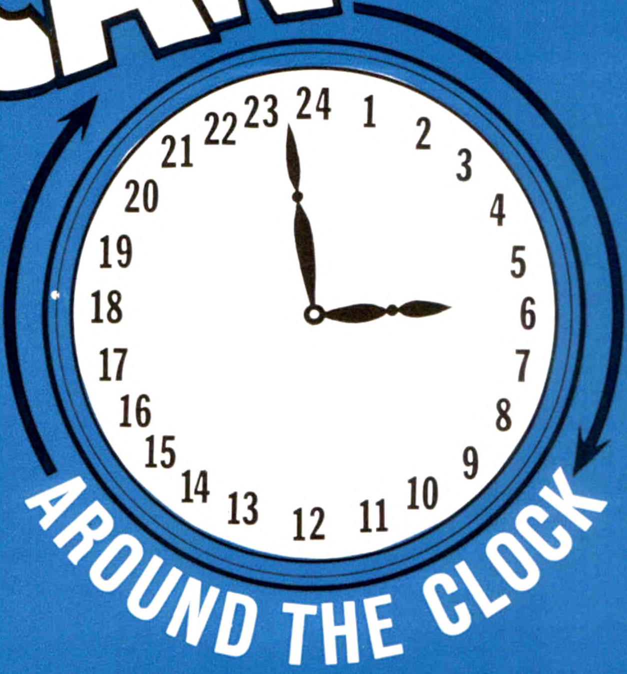

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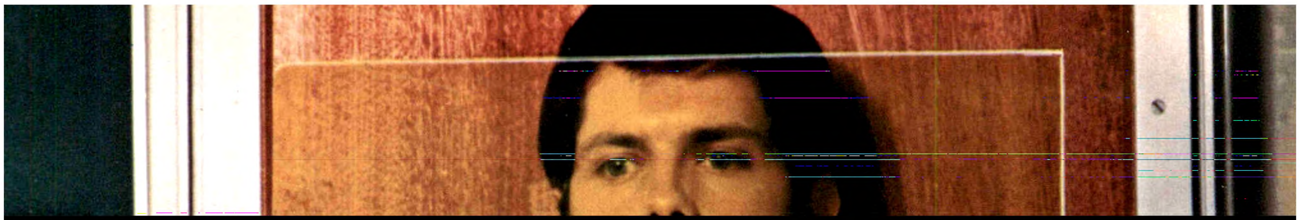

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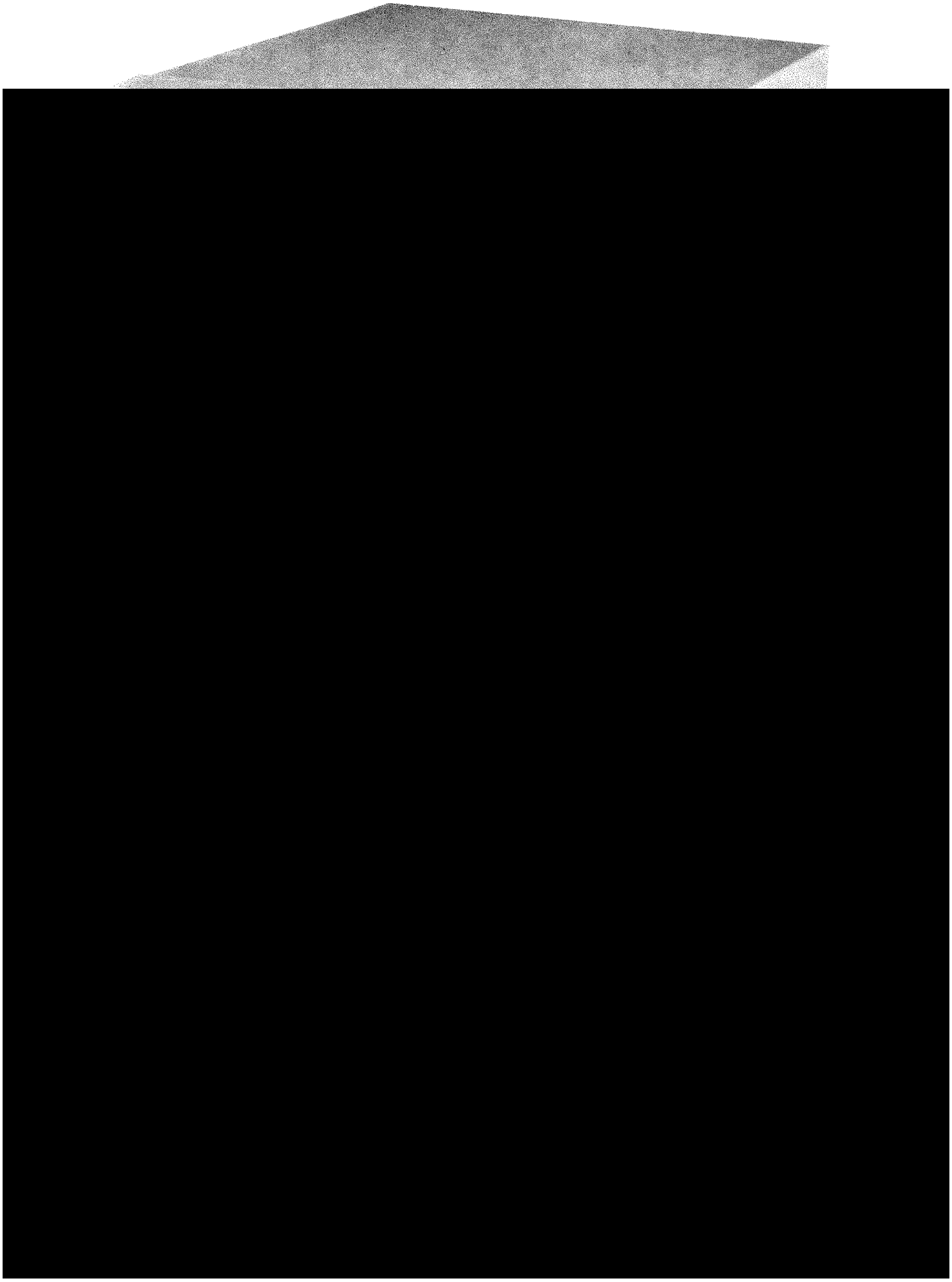

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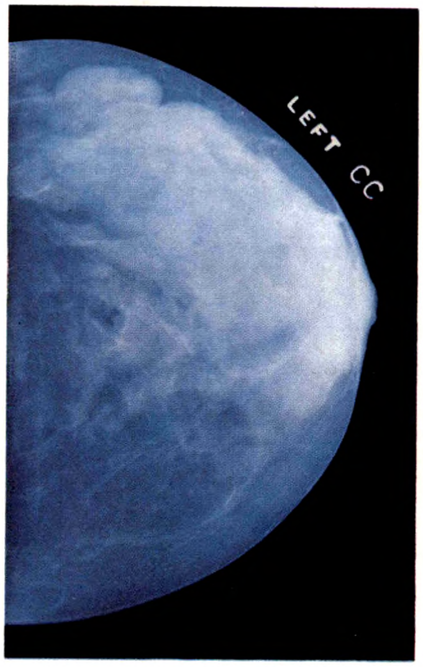

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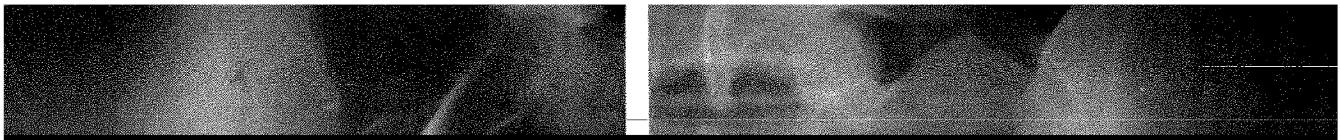

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THE AMERICAN JOURNAL

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Batches of the material have varied in specific activity from 300 to 500 microcuries per milligram of albumin. Thus, at a maximum, the patient receives no more than 10 milligrams of albumin. Other than occasional complaints of headache and nausea (which are commonly encountered even following diagnostic lumbar punctures and conventional myelographies), the patients suffered no ill effects from these studies.

Utilizing the Anger scintillation camera fitted with a 4,000 hole high resolution collimator designed for use with low energy gamma emitters, the patients were examined at 30 minutes, 2 hours, 4 hours, and 24 hours after injection. In addition to scintiphotos of the lumbar and cervical spine and the head, external scintillation count rates were also tabulated for each projection. Peripheral venous blood samples were obtained simultaneously and assayed for radioactivity in a well counter. To ensure accuracy and reproducibility of counting sites, positioning of the patient with respect to the crystal was checked on the cathode ray tube of the scintillation camera.

Unless the solution is introduced outside the subarachnoid space or later leaks out of that space through the puncture hole, all of the radioactivity in the early studies should be located in the cerebrospinal fluid, so that additional shielding of regions outside the spinal canal and head was not considered necessary.

However, leakage was a real problem in this series, despite monitoring of the needle position through the preceding myelography. Serial scintiphotos of the lumbar region demonstrated significant activity outside the subarachnoid space in 13 of the 54 patients (Fig. 1), preventing any meaningful quantitative studies on these 13 patients (Table I). Often, this leakage was not apparent on the 30 minute scintiphotos but only became evident on the 2 and 4 hour studies. The fact that leakage occurred in 24 per cent of this group of patients in whom extraordinary precautions were taken to ensure a complete subarachnoid injection of the radioactive material suggests that this extravasation is a real problem which may cause difficulties in interpretation of clinical studies performed without such precautions in the evaluation of patients with hydrocephalus.

TABLE I

SCINTICISTERNOGRAPHY IN NORMAL INDIVIDUALS

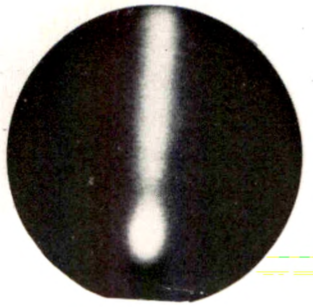
Total Patients Studied	54
Leakage Outside Subarachnoid Space	13
Ventricular Backflow	3
Patients Utilized for Quantitative Studies	38

For the quantitative studies, all regional external count rates were normalized to the posterior lumbar count rate at 30 minutes. Although mixing of the radioactive bolus with the cerebrospinal fluid occurs almost instantaneously with subsequent migration upward of the radioactivity within the spinal canal, physical limitations prevented evaluation by the scintillation camera earlier than 20 to 30 minutes after injection. As a result, the posterior lumbar count rate at 30 minutes after instillation was selected as the standard or baseline and all subsequent count rates were normalized to this value. The count rate over a given area was divided by the posterior lumbar count rate at 30 minutes, and the resulting percentage was termed the "count rate ratio." All count rates were corrected for decay prior to calculating this ratio. Technetium 99m has a half-life of 6 hours; at 24 hours, only 6 per cent of the original radioactivity remains. Thus, it is extremely difficult to carry these studies beyond 24 hours.

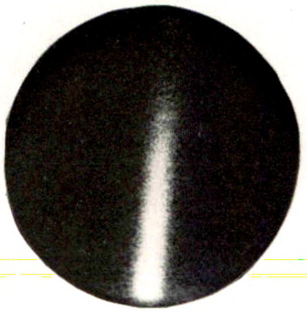
Activity in 2 cc. aliquots of peripheral venous blood was compared with the external count rate over the posterior cervical region at 2 hours, the resulting value being known as the "venous blood activity ratio."

RESULTS: THE NORMAL SCINTICISTERNOGRAM

Of the total of 54 studies in the identical number of patients, 38 demonstrated a composite visual picture which satisfied the criteria heretofore accepted as being



Post Lumbar



Post Cervical

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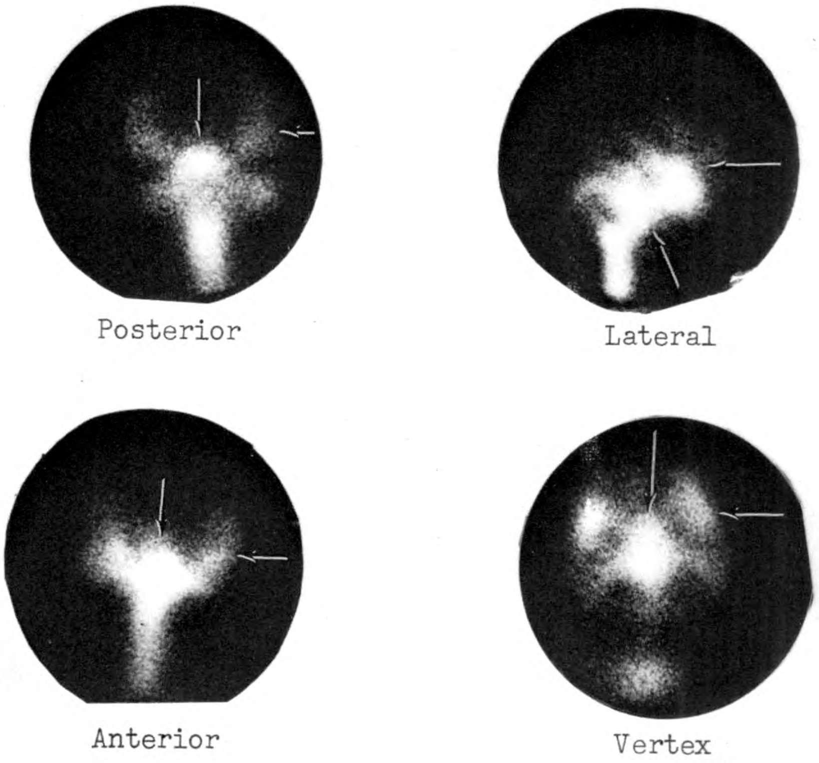


Fig. 3. Case 4. Normal scinticisternograms at 2 hours. The radioactive albumin has ascended into the basilar cisterns (vertical arrows), and some activity is already visualized in the circular sulci and sylvian fissures (horizontal arrows).

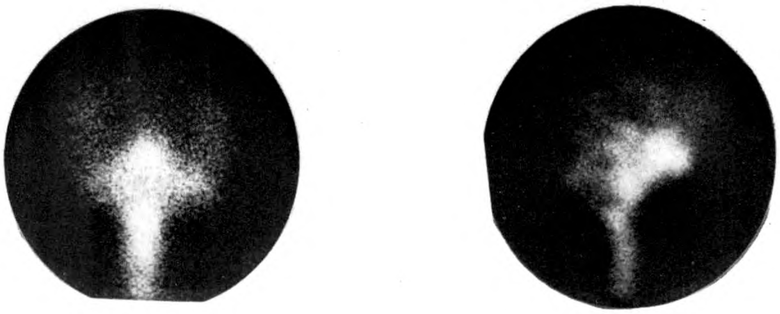
this table also indicates a wide range of values for each measurement. For example, in 9 cases the rate of progression of the radioactivity was considerably more rapid than in the group which closely approximated the mean values. A representative case in this group of 9 patients with rapid ascent is depicted in Figure 6. In this patient, the scintiphoto of the posterior head at 4 hours demonstrated considerable

Table II

distribution of intrathecal Tc^{99m} albumin

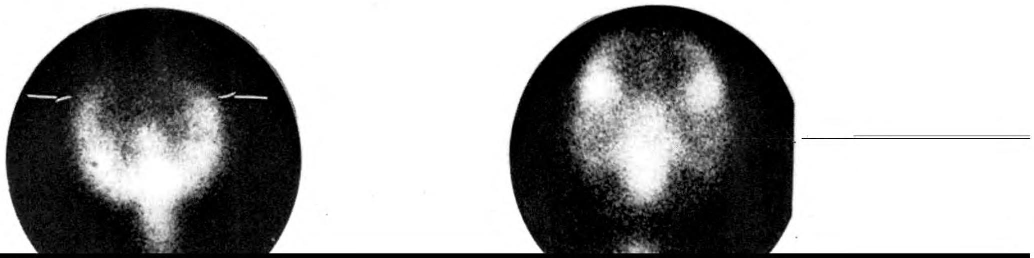
	Mean Count Rate Ratios (Per Cent) in 38 Normal Individuals*			
	30 min.	2 hr.	4 hr.	24 hr.
Posterior Lumbar	*100*	*69* (30–120)	*50* (9–89)	*43* (17–69)
Posterior Cervical	*18* (1–49)	*26* (9–78)	*24* (4–60)	*30* (12–107)
Posterior Head	*9* (1–42)	*23* (6–98)	*34* (6–103)	*61* (27–153)
Anterior Head	—	*26* (5–108)	*37* (6–116)	*64* (36–141)
Lateral Head	—	*30* (4–134)	*43* (14–128)	*75* (36–165)
Vertex	—	*25* (6–107)	*39* (10–153)	*78* (43–170)

* External count rates expressed as percentages normalized to the posterior lumbar rate at 30 minutes and corrected for decay. Figures in parenthesis represent the range of values in this group of 38 individuals.



Posterior

Lateral



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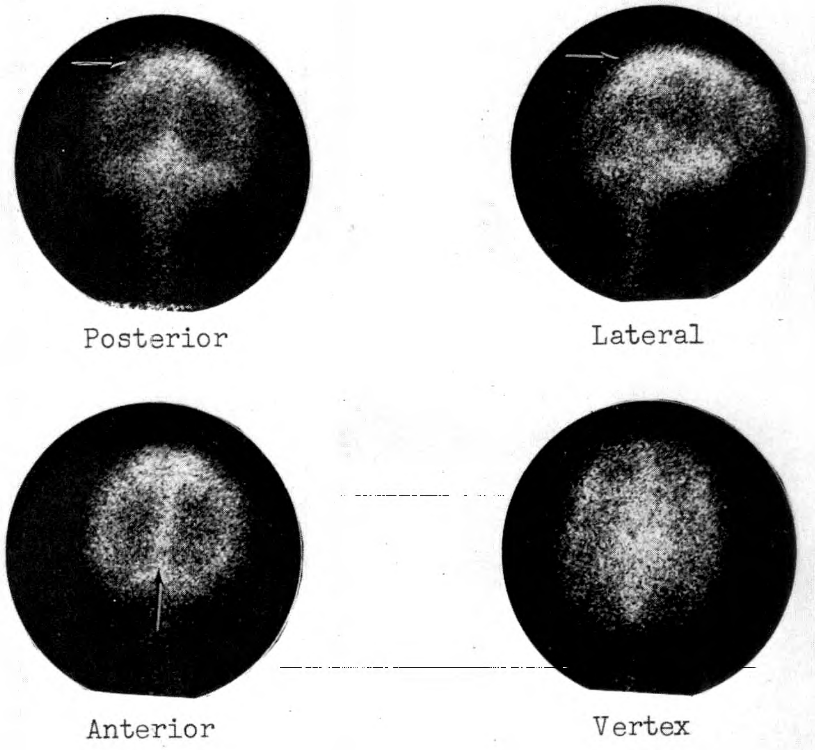

Fig. 5. Case 4. Normal scinticisternograms at 24 hours. The major portion of the activity is located high over the cerebral convexities (horizontal arrows) and in the midline longitudinal fissure (vertical arrow).

sumed by the patient during the course of the examination had no relation to this lateralization of flow. The remaining 25 patients showed a symmetric distribution of radioactivity over both convexities.

In addition to the series of 38 presumptive normal studies, 3 additional patients demonstrated early appearance of the labelled albumin in the cerebral ventricles on the 2 and 4 hour studies. In all 3 cases, this ventricular backflow occurred simultaneous with ascent of activity into the cerebral subarachnoid cisterns. By 24 hours, however, there was no activity identified pictorially in the ventricles in any of these 3 cases (Fig. 8).

Table IV

Intrathecal Tc^{99m} albumin studies—slow ascent

Case 6	Count Rate Ratio (Per Cent)			
	30 min.	2 hr.	4 hr.	24 hr.
Posterior Lumbar	100	83	70	32
Posterior Cervical	7	10	21	16
Posterior Head	1	6	14	49
Anterior Head	—	6	12	64
Vertex	—	7	10	75

The venous blood activity ratios reflect the absorption of the labelled albumin from the subarachnoid space into the bloodstream. The mean values for the group of 38 normal patients are listed in Table v. In general, these ratios were directly proportional to the external count rate ratios over the head, particularly the measurement made over the vertex. In particular, the venous blood activity ratio at 24 hours after injection showed a close correlation with the 24 hour external count rate ratio over the vertex. However, there were several exceptions to this rule. In the 3 pa-

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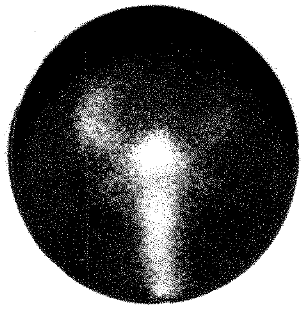
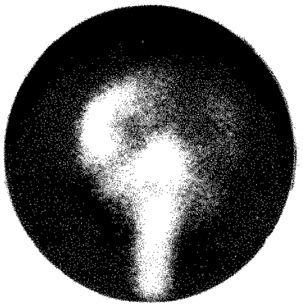
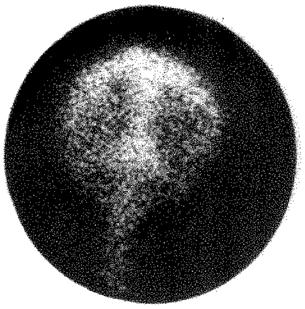


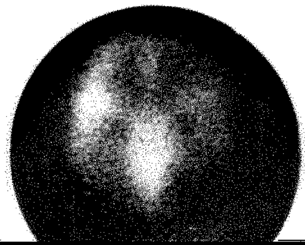
Posterior 2 Hrs.

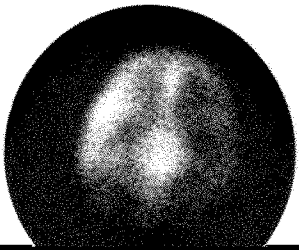


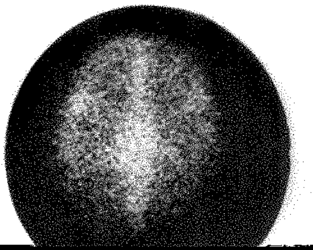
Posterior 4 Hrs.



Posterior 24 Hrs.







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et al.[9] have demonstrated the inaccuracy of hydrostatic pressure readings made through the lumbar puncture needle. Thus, scinticisternography has become, in the opinion of many investigators, the most valid test for the diagnosis of communicating hydrocephalus. It is therefore most important that the variations described in this group of presumably normal patients be included in the spectrum of normality. In particular, transient ventricular backflow appears to lie within that spectrum or at least somewhere short of the fully developed clinical syndrome of communicating hydrocephalus.

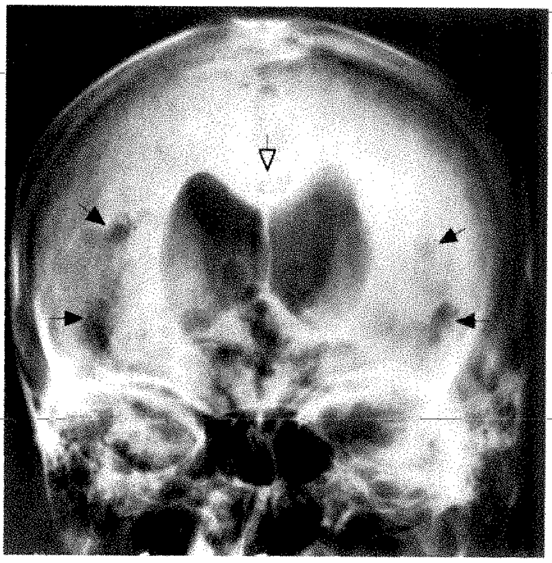

Fig. 9. Anteroposterior supine (brow up) projection of a pneumoencephalogram in a 61 year old male who on scinticisternography demonstrated transient ventricular backflow. The frontal horns and bodies of the lateral ventricles are markedly dilated. The circular sulci (closed arrows) and the pericallosal cistern (open arrow) are also enlarged, and no visualization of sulci above these regions could be obtained despite special effort.

The methods employed in this communication are by no means completely quantitative. The data presented are in the form of ratios relating activity over a given region of the cerebrospinal axis or in the peripheral venous blood to baseline count rates in the spinal subarachnoid space. Thus, an accurate statement of the exact proportion of the radioactive bolus in a given area at a given time cannot be obtained from these data. The external count rates, particularly in the 4 and 24 hour measurements, reflect activity not only in the cerebrospinal fluid but also in the blood, and we have no method to separate these two compartments. The venous blood activity ratio appears to offer a rough estimate of the degree of absorption of the labelled protein from the subarachnoid space into the blood.

Abbott and Alksne,[1] utilizing iodinated albumin (I^{125}), have attempted a more precise measurement of percentage absorption by multiplying the concentration of radioactivity in an aliquot of plasma by the plasma volume as determined by the RISA method. Their results in small groups of experimental animals, normal patients, and patients with communicating hydrocepha- [redacted]

accuracies of the RISA method of plasma volume determination. Furthermore, precise percentage absorption studies using technetiated albumin would be even less accurate since technetiated albumin is cleared from the blood stream much more rapidly than iodinated albumin. Thus, it would seem advisable to avoid using the plasma volume in these calculations, but rather obtain a ratio relating activity in the spinal subarachnoid space early in the examination, when absorption should be minimal, to the activity in the venous blood at 24 hours. The relatively low venous blood activity ratios in patients with transient ventricular backflow (Table v) suggest that there is diminished absorption of al-

CONCLUSIONS

1. Quantitative scinticisternographic studies in a group of 54 presumably normal individuals have established both a mean and a range of normal for this examination.

2. Significant variations from what has heretofore been accepted as normal have been found in this group. These include variations in the rate of ascent and absorption of the labelled albumin, frequent lateralization of flow to one or the other side intracranially, and occasional transient backflow into the cerebral ventricles. Leakage outside the subarachnoid space at the site of lumbar puncture was frequent, preventing quantitative studies in 13 patients (24 per cent of the total group).

3. Scinticisternography, including both pictorial and quantitative examinations, is a valid diagnostic test in the evaluation of cerebrospinal fluid dynamics in normal and hydrocephalic patients. The range of nor-

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VENTRICULO-VENOUS CEREBROSPINAL FLUID SHUNTS[*]

ROENTGENOLOGIC ANALYSIS

By JEREMY ALTMAN, M.D.,[†] *and* A. EVERETTE JAMES, JR., M.D.[‡]
BALTIMORE, MARYLAND

THERE has been recent increased interest and technical improvement in treatment of hydrocephalus by ventriculo-venous shunts. Radiologists should be familiar with the normal roentgenologic appearance of these shunts and the manifestations of attendant complications. Isolated reports describing several commonly used valve systems have appeared in the literature, but no comparison of various valve systems and emphasis on their roentgenologic features could be found.[7,16] The purpose of this communication is to characterize the most common ventriculo-venous shunts emphasizing the roentgenologic features useful in assessing complications and reasons for therapeutic failures.

There are 3 ventriculo-atrial valve systems commonly in use for the treatment of hydrocephalus. Each maintains unidirectional diversion of cerebrospinal fluid (CSF) from the lateral cerebral ventricle to the cardiac right atrium. In each shunt system there is a ventricular catheter, one or more unidirectional valves, a flushing device and a cardiac catheter.

VALVE SYSTEMS

I. HOLTER VALVE SYSTEM

In 1956, John Holter designed a unidirectional valve system for release of excess ventricular fluid into the blood stream. The system consists of[8] (Fig. 1, *A–C*):

1. A radiopaque right angle catheter made of silicone rubber for cannulating the ventricular system. The catheter tip has multiple small side holes for influx of CSF.
2. A valve assembly consisting of 2 condom or "fish mouth" check valves of silicone rubber housed in stainless-steel jackets and linked by flexible tubing.
3. A cardiac catheter made of radiopaque silicone rubber tubing connected to the distal end of the valve assembly.

The use of silicone rubber in the valve assembly is important because of its properties of self-adherence. This adherence allows the slit-like orifice in the valve cap to open at constant pressure and close as soon as CSF pressure falls.[17] Silicone rubber does not lose its elasticity; its resistance properties remain intact for as long as 8 years after implantation.[4] Another property of silicone rubber is that it produces minimal tissue reaction. Thrombus formation is also discouraged by its nonwettable surface.[24]

The Holter valve is designed so that the system's patency can be tested by compressing the tubing linking the valves against the skull. Normally, fluid can be easily forced through the outlet valve and cardiac catheter. Following compression, the tubing will resume its normal shape when fluid is drawn in from the ventricular catheter and inflow valve. Inability to compress the tubing indicates obstruction in the system's distal limb; failure of the tubing to resume its normal shape after compression indicates obstruction in the proximal limb.

The cardiac catheter of the Holter valve is threaded through the common facial vein

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[*] Presented as a Scientific Exhibit at the Seventy-first Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 29–October 2, 1970.

From the Department of Radiology, The Johns Hopkins Medical Institutions, Baltimore, Maryland.

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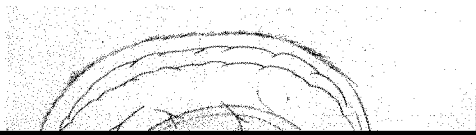
[‡] Advanced Academic Fellow of the James Picker Foundation on recommendation of the Committee on Radiology, National Academy of Science, National Research Council. Department of Radiological Sciences, The Johns Hopkins School of Hygiene and Public Health, Baltimore, Maryland.



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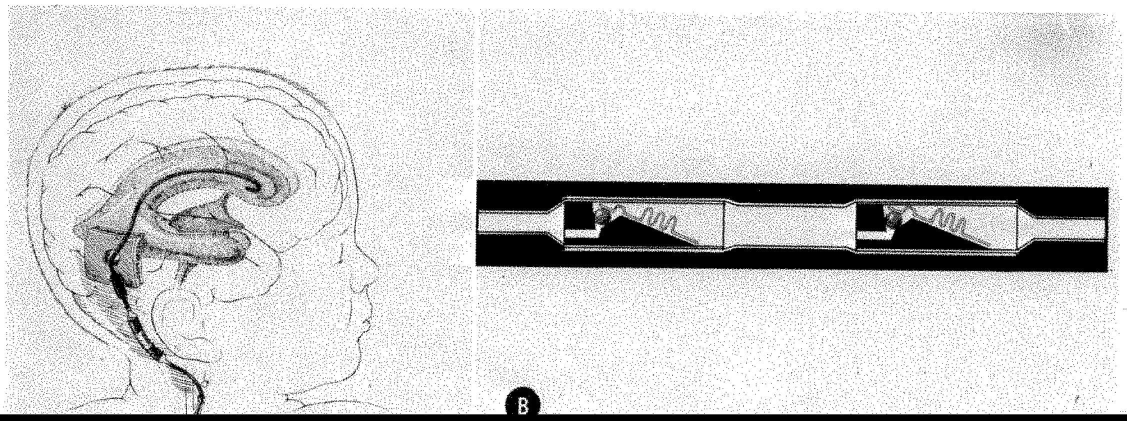

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TABLE I

VALVE SYSTEMS

Type	Ventricular Catheter	Reservoir	Valve	Connectors
Holter	straight	metallic, in burr hole (optional)	twin "fish mouth" valves in metallic housing	—
Pudenz-Heyer	straight	silicone rubber, with flushing device in burr hole	slits in distal atrial catheter	nylon
Hakim	"shepherd's crook" curve	silicone rubber antechamber proximal to valve (optional)	twin ball valves in metallic housing	metallic, between ventricular catheter and antechamber

It connects to the ventricular catheter and a tube leading to the valve. The Holter valves are metal-encased and connected by a short length of tubing. They are characteristically placed in a subgaleal tunnel behind the mastoid area, but may be located in parietal or occipital areas. The cardiac catheter is radiopaque and descends in the right internal jugular vein and superior vena cava to the region of the right atrium. Type A ventricular catheters have a constant diameter; Type B and C taper distally. The Holter system can be identified by its radiopaque ventricular catheter, twin valve housings of uniform metallic density connected by a short tube behind the mastoid area, and radiopaque cardiac catheter.

The Pudenz-Heyer assembly is characterized by a straight ventricular catheter that is radiopaque only at its tip (Fig. 5*A*; Table 1). This catheter connects to the

[redacted]

Hakim apparatus (Fig. 6*A*; Table 1). A small metallic cylinder connects the ventricular catheter to the radiopaque antechamber. The valve is similar in roentgenographic appearance to the Holter system but the characteristic oblique distal end and ball seat can often be recognized. Metallic twin valves are linked by a short connecting tube and usually placed behind the mastoid area. The cardiac catheter is radiopaque and descends in the right internal jugular vein and superior vena cava to the region of the right atrium (Fig. 6*C*). In identification of the Hakim apparatus, the radiopaque curved ventricular catheter connected to the antechamber, or valves connected by a small metallic cylinder are the most characteristic features.

These valve systems have several basic components in common. However, the radiologist can identify which valve has been utilized by the configuration and radioden-

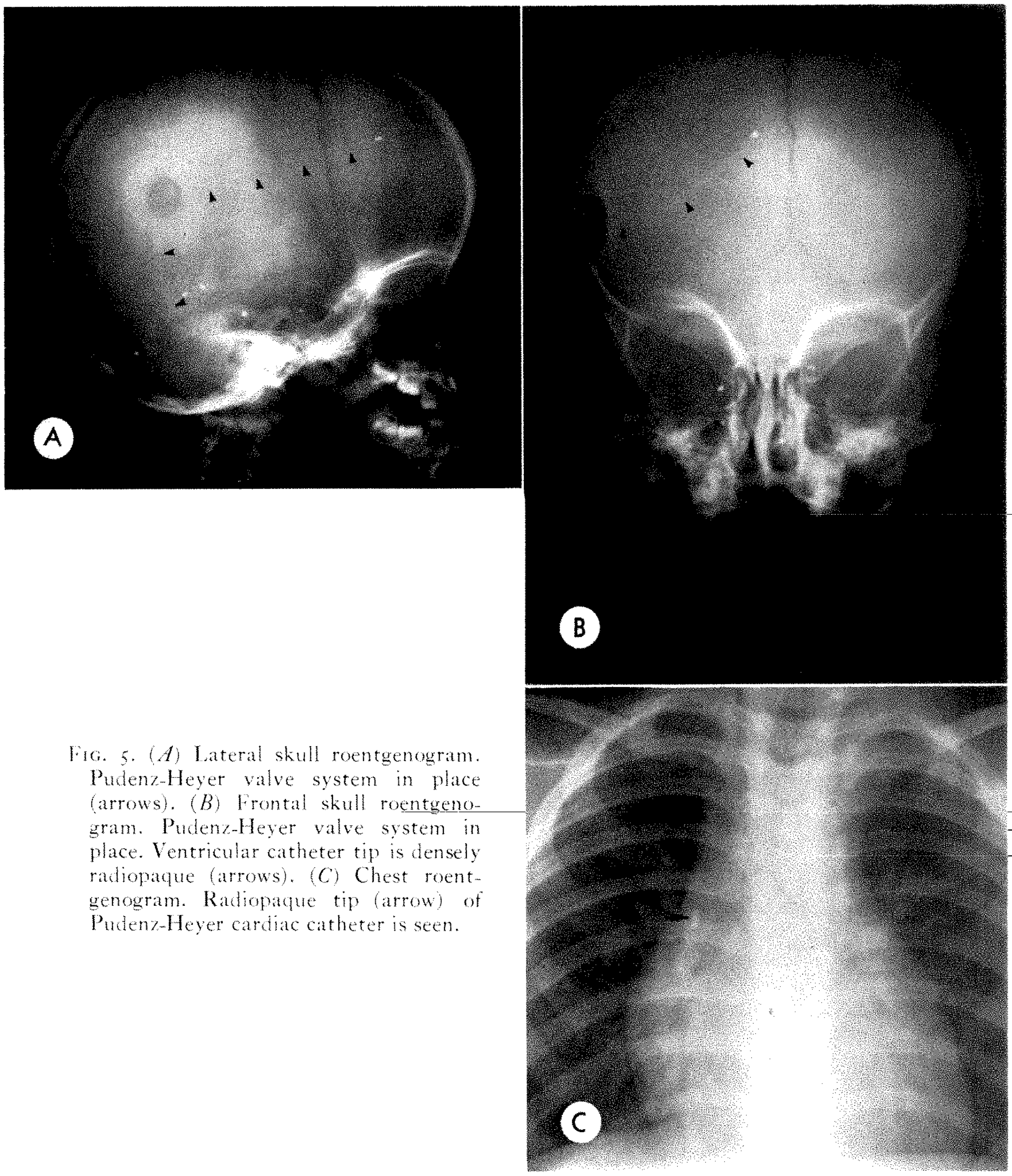


Fig. 5. (*A*) Lateral skull roentgenogram. Pudenz-Heyer valve system in place (arrows). (*B*) Frontal skull roentgenogram. Pudenz-Heyer valve system in place. Ventricular catheter tip is densely radiopaque (arrows). (*C*) Chest roentgenogram. Radiopaque tip (arrow) of Pudenz-Heyer cardiac catheter is seen.

The position of the ventricular and atrial catheters is very important in the roentgenographic assessment. The tip of the ventricular catheter should be in the frontal horn of the lateral ventricle.[19] On the lateral skull roentgenogram, the radiopaque tip should be anterior to the coronal suture.

The radiopaque tip of the cardiac catheter is usually positioned at the level of the inferior aspect of the sixth thoracic vertebra on chest roentgenograms taken with portable unit at the time of the operation. Roentgenographic contrast material may be instilled into the catheter to improve visualization during insertion. Another method is to fill the catheter with

[redacted]

saline and use it as an electrode for placement by intracardiac electrocardiography.[31]

In addition to examination for catheter position and physical integrity of the system, introduction of a radiopharmaceutical into the CSF space to image CSF dynamics (cisternography) can be utilized to assess function. A radiopharmaceutical may be placed directly into the shunt and images obtained along the shunt pathway and over the distal catheter tip in the right atrium. To evaluate the entire shunt system in patients with communicating hydrocephalus, a radiopharmaceutical may be injected intrathecally in the lumbar area and its course followed from the lateral ventricles into the right cardiac atrium. In studies of this type, the radiopharmaceutical should not diffuse across the CSF membrane during the period of the study and must have an appropriate effective half life (I^{131}HSA, Tc^{99m} albumin, Yb^{169} DTPA have been used in our laboratory). In patients with noncommunicating hydrocephalus, the radiopharmaceutical may be injected directly into the lateral ventricles.

DISCUSSION

The most common clinical use of the various shunt systems is in treatment of hydrocephalus. Hydrocephalus refers to any condition in which there is an excess of cerebrospinal fluid in proportion to the cerebral volume. Two general categories of hydrocephalus with increased pressure are recognized:[8]

1. Obstructive, in which there exists a mechanical block at some point between the lateral ventricles and the outlet foramina of the fourth ventricle.
2. Communicating, in which no obstruction to the flow of cerebrospinal fluid in the ventricular system is found.

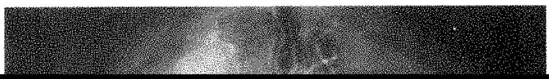
In addition, an occult form of hydrocephalus with CSF pressure in the normal range but different from that associated with brain atrophy or degeneration has been characterized by Adams *et al.*[1]

Therapeutic approaches have consisted of the extirpation and obliteration of the choroid plexus, a major contributor to the formation of cerebrospinal fluid (choroid plexectomy), restoring the communication between producing and absorbing structures (third ventriculostomy, ventriculocisternostomy), diversion of cerebrospinal fluid into extracranial spaces, including the mastoid, ureter, pleural cavity, peritoneal cavity, subdural space, and shunting CSF into the circulatory system.[29] In the past two decades, major advances have been made in the development of neurosurgical shunts to divert CSF from the lateral cerebral ventricles to the cardiac right atrium. The number of patients treated with these shunts has greatly increased.

Gartner, in 1895, suggested that the ventricles in hydrocephalus could be decompressed by shunting cerebrospinal fluid into the vascular or lymphatic compartment. Payr, in 1908, first connected the lateral ventricle with the internal jugular vein, and reported successful decompression in 3 of 8 cases.[8] During the next 30 years, various investigators met with little success in attempting to establish shunts to the systemic circulation until the work of Ingraham *et al.*[14] in 1948. Hydrocephalus produced experimentally in dogs was treated with a polyethylene shunt tube connecting the lateral ventricles to the sagittal sinus or superior vena cava. In the anesthetized state, the system functioned well, but obstruction of the catheter due to blood clot occurred when the dogs awakened. Ingraham *et al.* concluded that a unidirectional valve would be necessary to prevent reflux of blood into the shunt during episodes of increased intrathoracic pressure.

In 1952, Nulsen and Spitz[20] reported the first successful artificial valve used in a shunt system. Two unidirectional stainless-steel ball valves connected by a rubber pump prevented backflow in a system that diverted CSF from the lateral ventricle into the superior vena cava. Holter modified the Spitz design in 1956, substituting silicone rubber "fish mouth" valves.[8]

Ventriculo-venous shunting offers numerous advantages in the palliation of hydro-



may provoke acute pulmonary edema and congestive heart failure. The sudden de-

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HYPOPLASIA OF THE INTERNAL CAROTID ARTERY ASSOCIATED WITH CEREBRAL PSEUDOANGIOMATOSIS

REPORT OF 4 CASES

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A PARTICULAR variety of cerebrovascular anomaly, consisting of hypoplasia of the internal carotid artery (often bilateral) with narrowing at the level of the supraclinoid portion of its siphon and with an unusual pseudoangiomatous pattern of the cerebral circulation almost resembling an "arterio-arterial angioma" has been recently described.[5,10,12,13] Since all patients

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of the cerebral circulation on that side, together with angiographic signs of a left temporal space-occupying lesion which was interpreted as an intracerebral hematoma.

Improvement of the patient's neurologic status began shortly after admission and continued rapidly during the following days; a surgical procedure was, therefore, deemed unnecessary.

Upon discharge a couple of weeks later, the

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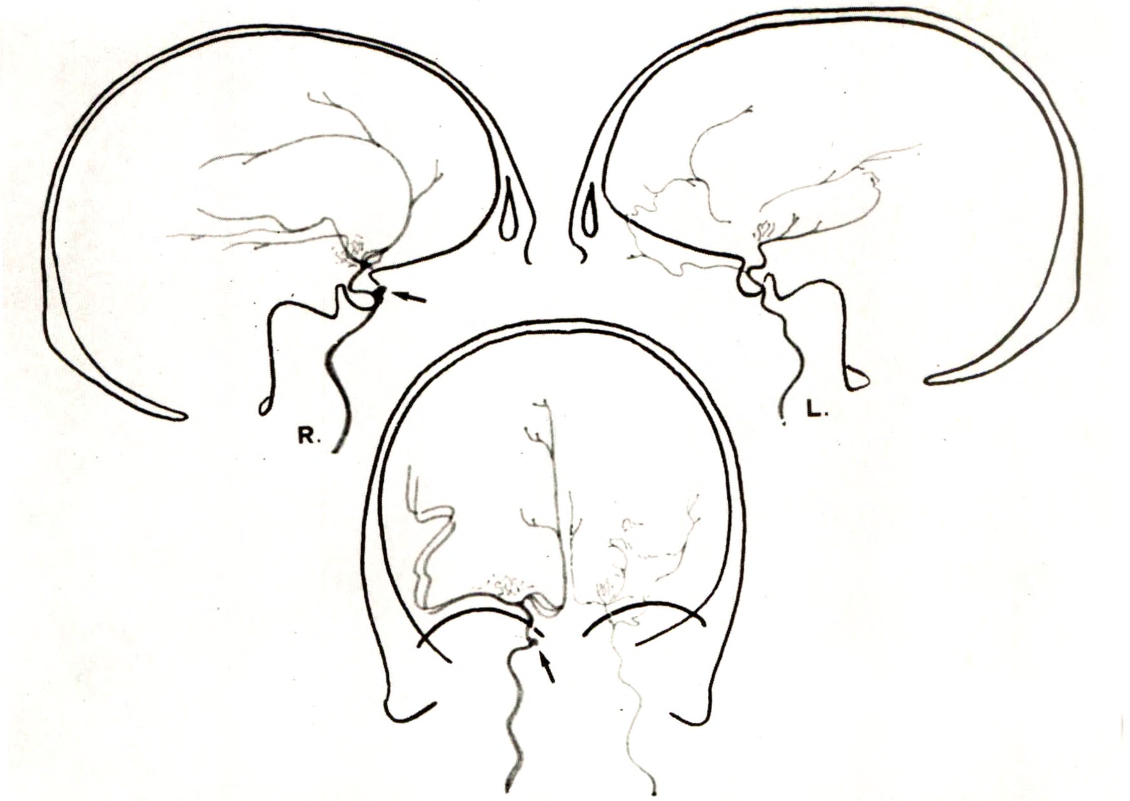


FIG. 2. Case I. Schematic representation of the arteriographic findings shown in Figure 1. The replenishment of the anterior cerebral artery from vessels originating from the ethmoidal branches of the ophthalmic artery was obtained in earlier phases than those shown in Figure 1.

sive mental confusion, dysphasia, and right-sided paresthesias which eventually led to her admission.

Upon neurologic examination, she exhibited a marked mental confusion and temporal-spatial disorientation, expression paraphasias, central deficit of the right facial nerve, right hemiparesis prevalent in the upper limb, impairment of epicritic sensation and loss of stereognosis on the right side of her body.

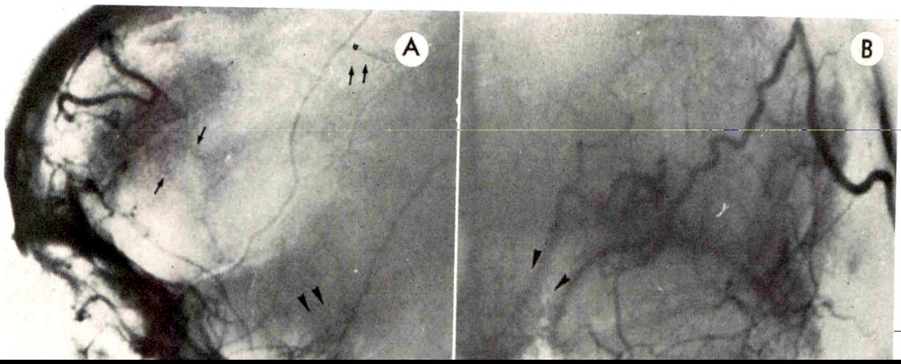
Electroencephalogram, brain scanning and lumbar puncture gave normal findings. Bilateral carotid arteriographies were performed (Fig. 5, *A–D*; and 6).

After treatment with vasodilating drugs, the patient made a complete recovery and was discharged.

CASE IV. P.L., a 4 year old girl who had been delivered 15 days after expected time, showed at birth a severe cyanosis and had to be given an exchange transfusion because of Rh factor incompatibility. During the following years, her physical growth was normal but her mental development was severely delayed: she suffered a marked oligophrenia and frequent temper tantrums which made her a behavior problem within the family and at kindergarten. Several times she had suffered minor head injuries without any apparent serious consequences; only 2 weeks prior to admission, following a fall, she suffered a clonic Jacksonian seizure affecting her right face and upper limb and followed by a residual right hemiparesis.

Upon examination, she showed a total inability to speak and to understand, and a marked right hemiparesis. An electroencephalogram revealed a left hemispheric lesion with involvement of the deep cerebral structures. A left carotid arteriography (Fig. 7, *A–C*) was done, followed by right cerebral arteriography via a retrograde injection into the brachial artery (Fig. 8, *A–D*; and 9).

She was discharged in unchanged condition



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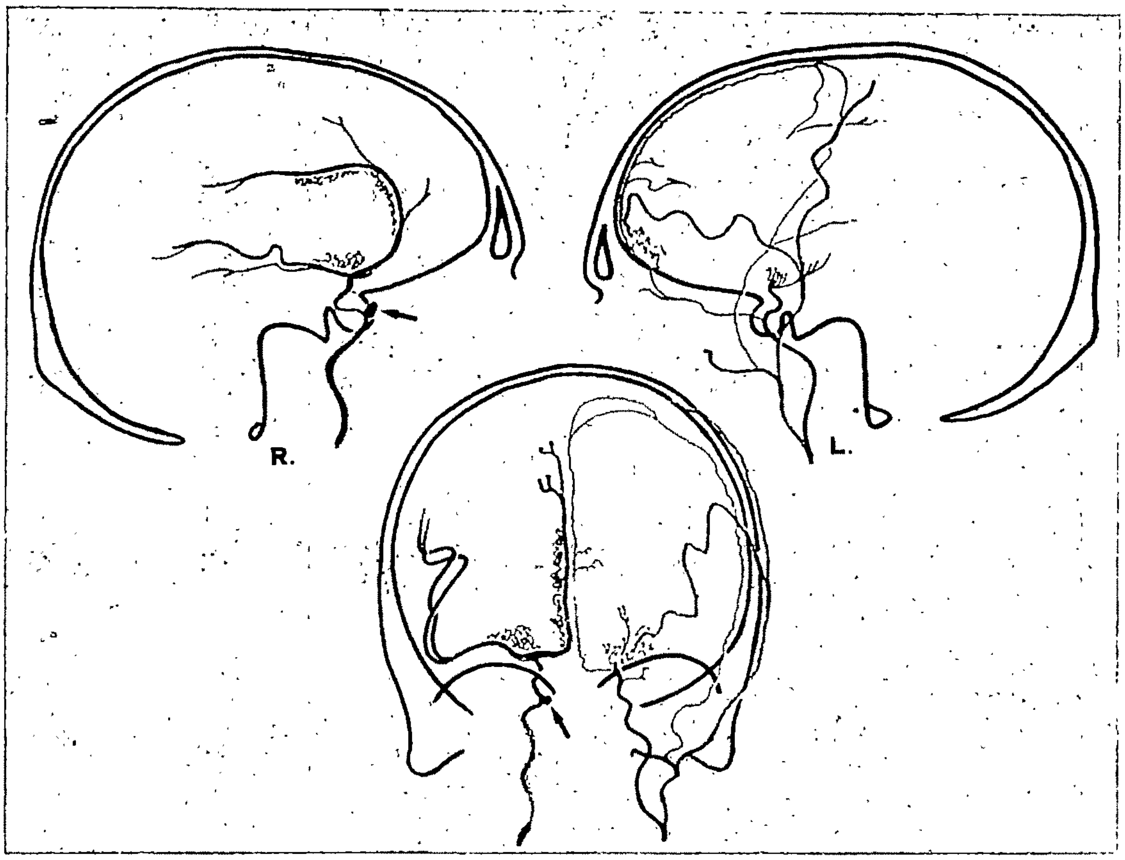

FIG. 4. Case II. Schematic representation of the arteriographic findings illustrated in Figure 3.

and, 1 year later, re-admitted for a control left carotid arteriography (Fig. 10, *A* and *B*). A control electroencephalogram showed no significant change from the one performed 1 year earlier.

DISCUSSION

There are 3 aspects of the angiographic findings of our patients which should be considered separately.

1. *Hypoplasia of the internal carotid artery with complete stenosis at the supraclinoid tract of the siphon.* The malformative origin of this abnormality appears to be proved, in our opinion, by its extension to, and by its uniformity through, the full length of the vessel—a fact which should rule out an acquired disease such as arteritis. The findings of Brihaye and Dhaene,[1] Priman and Christie,[14] and Van der Zwan and Fossen,[17] which are quite similar to those observed in our patients, would appear to confirm this hypothesis.

2. *The abnormal, pseudoangiomatous pattern with its peculiar features,* located at the base of the brain near the bifurcation of the internal carotid artery, has been interpreted[12,18] as a residual of the "rete mirabile" which is characteristic of some mammalian species. In fact, in some animals (cat, goat, sheep, ox and pig), in which blood is supplied to the anterior part of the brain through numerous, thin arterial vessels in various forms of anastomoses and intertwining, originating from branches of the external carotid artery (anastomotic branch and artery from the internal maxillary artery, ascending pharyngeal and accessory meningeal arteries), a real and true "rete mirabile" is present. Daniel *et al.*[2] and Lie[9] stated that the degree of development

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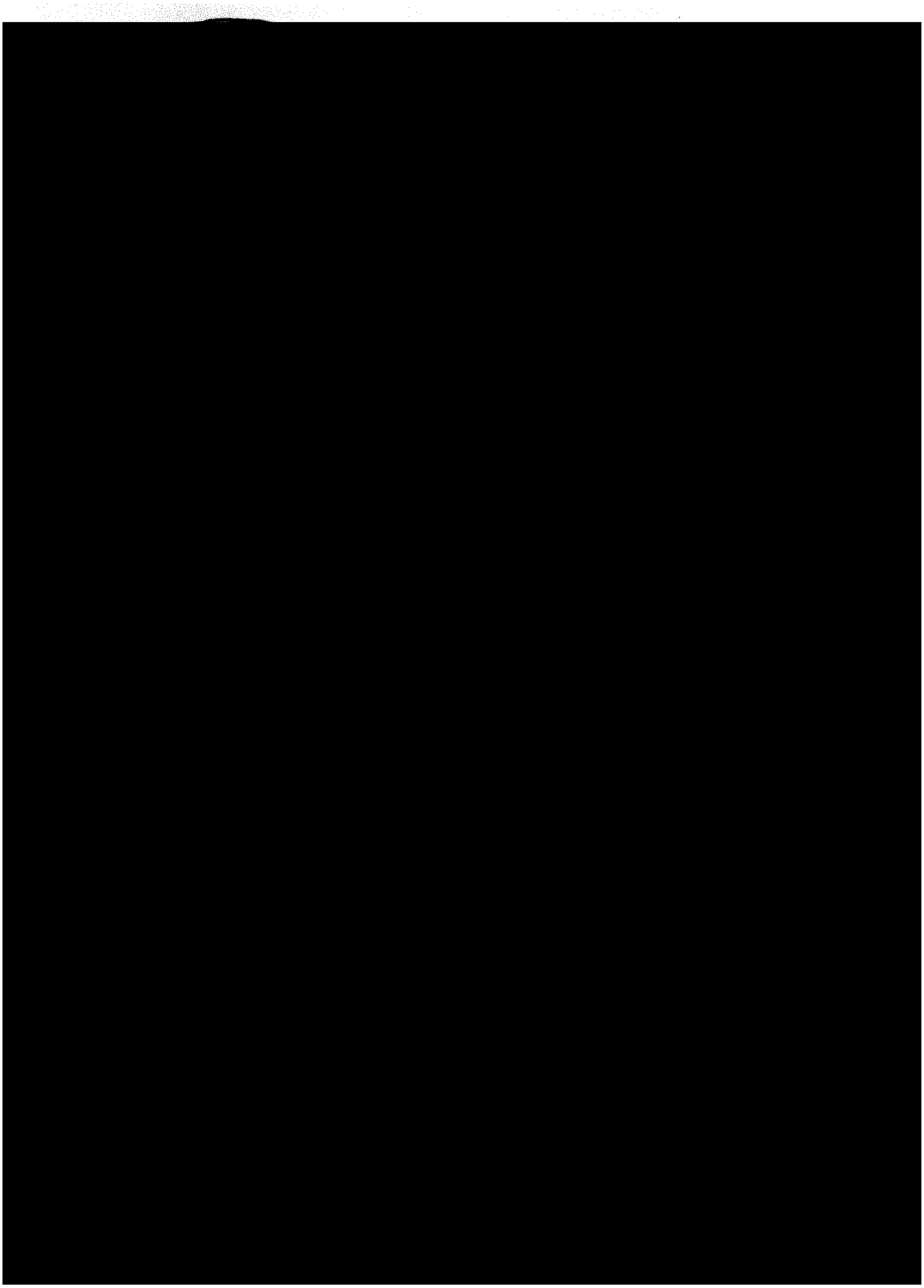

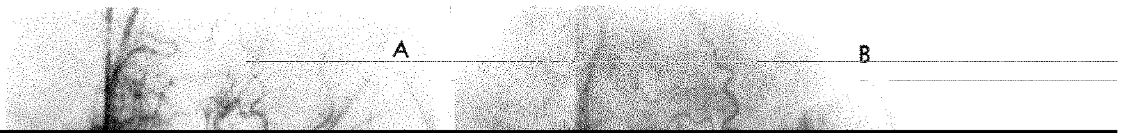

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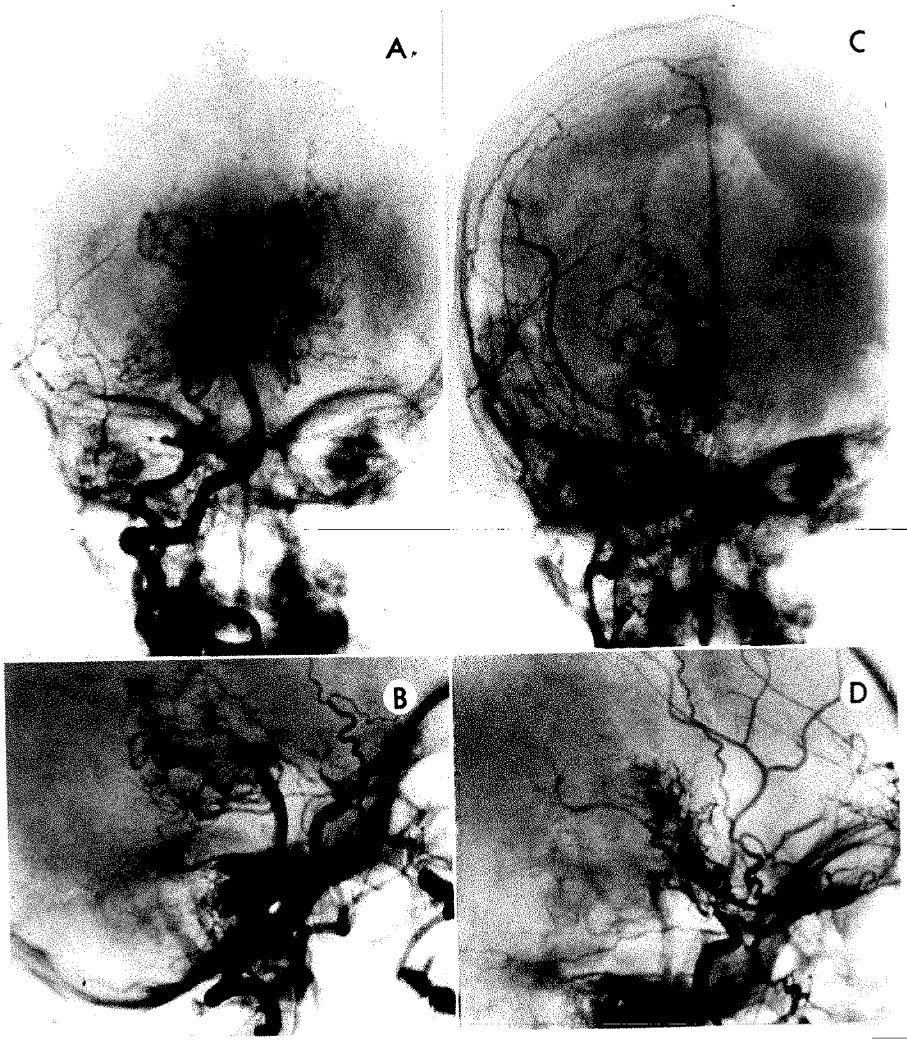

FIG. 8. Case IV. Right cerebral angiograms, via retrograde injection into the brachial artery, show a similar pattern in the right carotid circulation and in the vertebro-basilar district. There is a maximum filling of the basilar artery and its branches demonstrated in the anteroposterior (*A*) and lateral (*B*) views. There is a dense pseudoangiomatous network supplied by branches of the 2 posterior cerebral arteries, mainly by the posterior choroidal arteries. There is no evident tendency to replenishment of the supratentorial vessels. The internal carotid artery is still scarcely filled but a severe stenosis of its supraclinoid tract is already visible. (*C* and *D*) A pseudoangiomatous pattern with the same characteristics can be seen in a deep, paramedian location, immediately above the carotid artery stenosis. There is also a thick network in the territory of the ophthalmic artery and from the ethmoidal branches.

circulation along the carotid siphon. Other authors[10,12,16] support a congenital and malformative origin of this disease.

On the basis of the information obtained in our cases, we are inclined to support this latter viewpoint.[2] In 2 of our cases (Cases I and II), alterations of a definitely malformative origin were present (the 2 saccular aneurysms and the abnormality of the A/1 tract of the anterior cerebral artery). Further evidence is furnished, in our opinion, by the angiographic appearance of the left internal carotid artery in our first and second patients and of both internal carotid arteries in our third patient, with a uniform narrowing of the caliber, beginning at their origin and without any of the segmental narrowings or marginal irregularities which

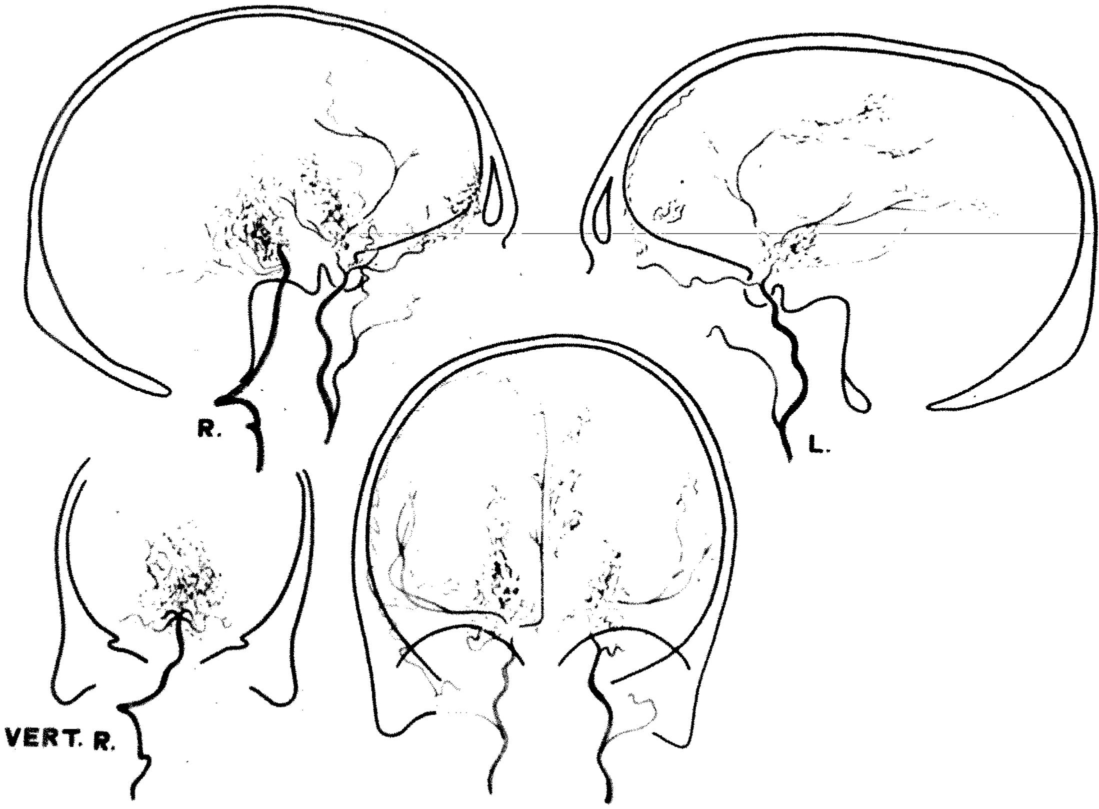

FIG. 9. Schematic representation of the angiographic findings illustrated in Figures 7 and 8.

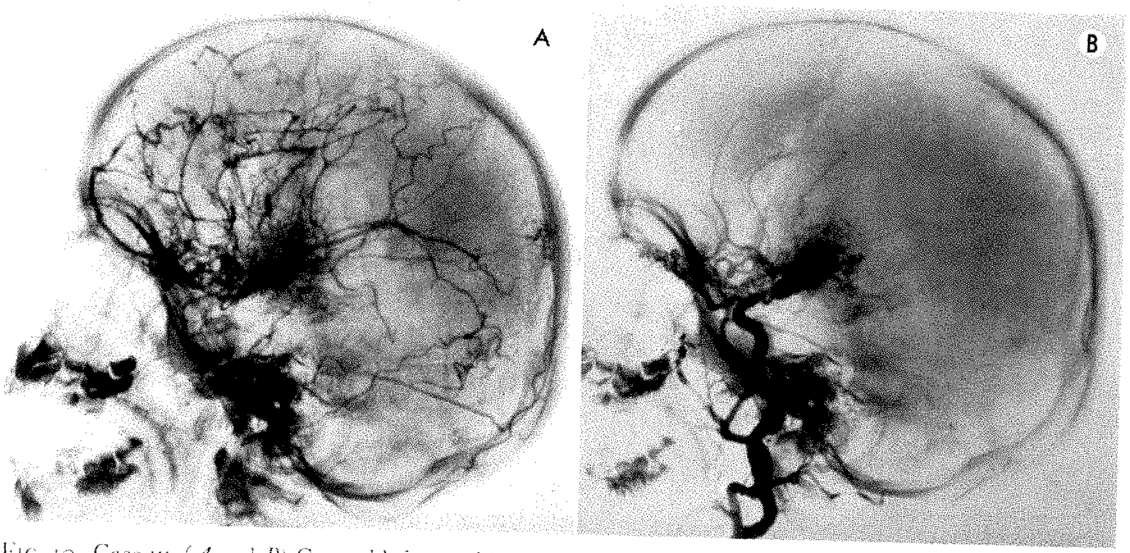

FIG. 10. Case IV. (*A* and *B*) Control left carotid arteriograms, 1 year later.

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are characteristic of arteritis or of acquired arterial thromboses.

The pseudoangiomatous circulation located along the carotid siphon which, according to supporters of the acquired origin of the disease, has the significance of collateral circulation originating from the anterior choroidal artery, presents such characteristics as to exclude both dependence on the anterior choroidal artery and functional efficacy as a collateral circulation.

It should be further noted that, contrary to the patients reported by Maki and Nakada,[10] and by Kudoh,[5,6] the disease in our cases has presented no tendency to progression, as proved by the arteriographic control studies and by the continuous clinical follow-up examinations.

The fact that symptoms of intermittent cerebral ischemia did not appear in our first 3 cases until adult age is, in our opinion, insufficient evidence against a malformative etiology. It is conceivable that the abnormality of the cerebral circulation becomes clinically evident only when progressive cerebro-vascular changes, compatible with adult life, result in a diminution of the functional reserves of the intracranial circulation. It should be remembered, furthermore, that the isolated autopsy reports so far available[4] have given no substantial contribution to a better knowledge and understanding of this condition.

The hypothesis of a malformative cause of this vascular abnormality is thus supported, in our opinion, by the association of other aspects which are clearly of congenital origin, by the characteristics of the stenosis of the internal carotid artery, by the features of the pseudoangiomatous pattern and by the lack of progression of the disease. However, some reservations must be kept in mind because of the complexity of all these angiographic patterns and the several differences found in the cases hitherto reported in the literature. It is possible, for instance, that different etiologic elements may coexist in this condition, or that the functional insufficiency of the carotid artery may eventually lead, in due time, to collateral circulation patterns quite peculiar and unusual. A better explanation to all these factors will be possible only after further clinical, angiographic and autopsy observations.

SUMMARY

Four cases of a peculiar cerebral vascular abnormality, consisting of hypoplasia and stenosis of the internal carotid artery, often bilateral, and of an unusual pseudoangiomatous cerebral circulation, are reported.

Such a condition, originally found in Japanese patients only, has an undetermined cause.

The information obtained through clinical and angiographic studies in these 4 cases seems to favor the hypothesis of a congenital and malformative origin of this vascular abnormality.

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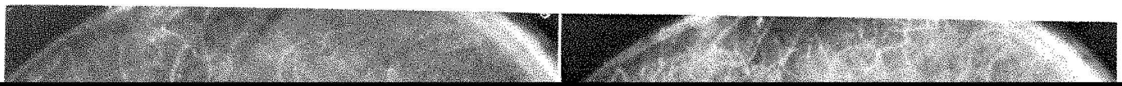

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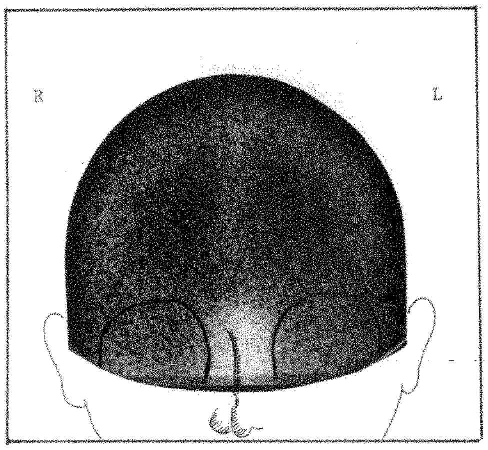

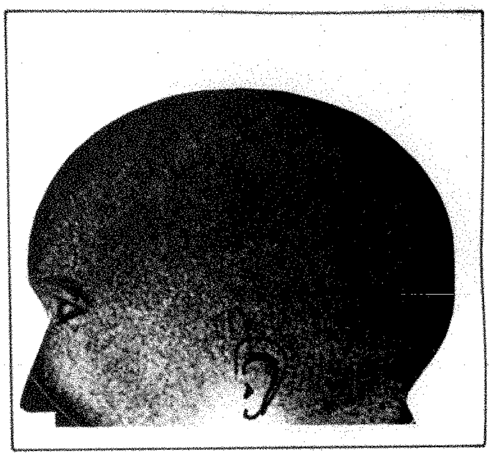

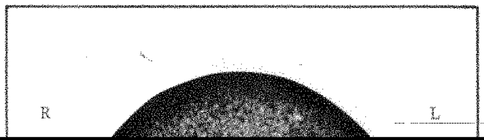

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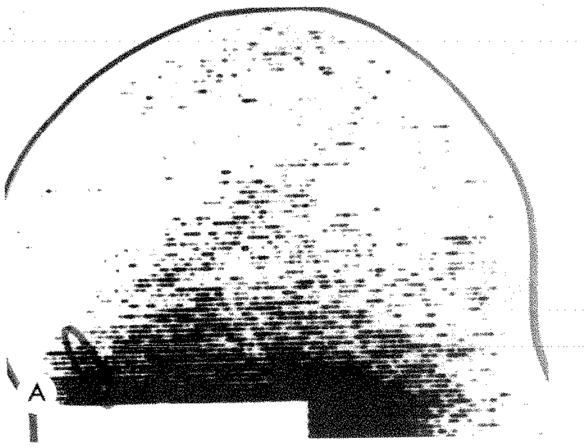
showed infiltration with neutrophils and monocytes and necrosis of vessel walls. Astrocytes and microglia were increased, and there was demyelinization of adjacent fiber tracts. No definite intranuclear inclusions were identified.

Comment. The fulminating course of the patient's illness, the temporal lobe mass at angiography, and the gross microscopic findings at postmortem examination are all consistent with a herpes simplex encephalitis. The virus was not isolated, so the diagnosis is presumptive in this case.



Case iv. A 64 year old man developed a shuffling gait and 3 days before admission was

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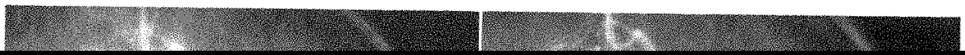

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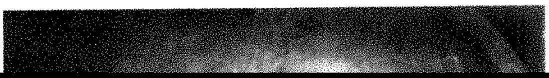
quently affected. Haymaker *et al.*[13] in their analysis of 37 cases also found necrosis in



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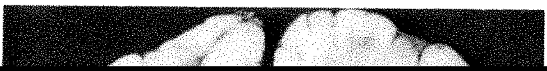

serologic tests are unreliable.[15] In a patient

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Table I

SUMMARY OF FINDINGS IN 4 CASES OF NECROTIZING ENCEPHALITIS

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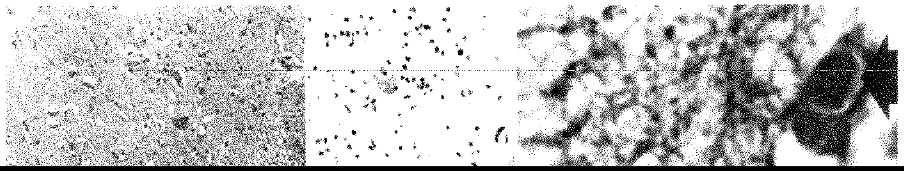

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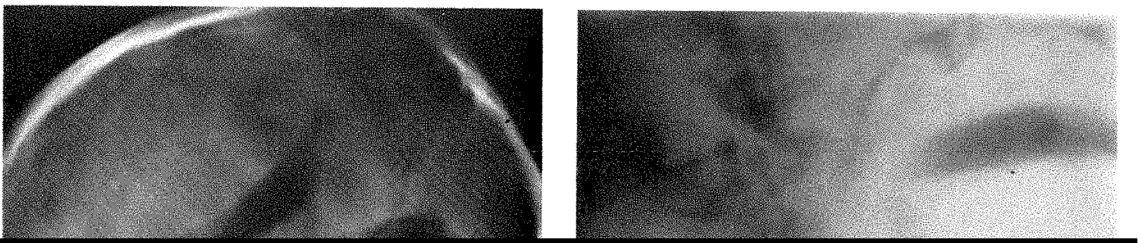

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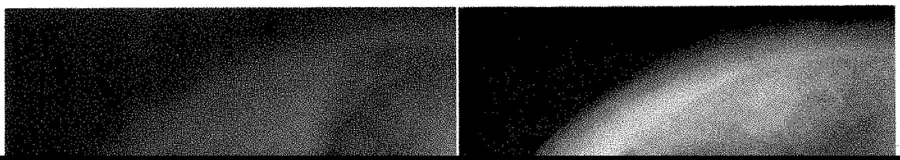

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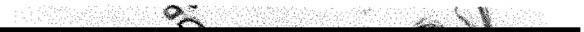
proximal portions of the left anterior and the



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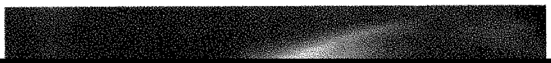

optic nerve was displaced laterally and su-

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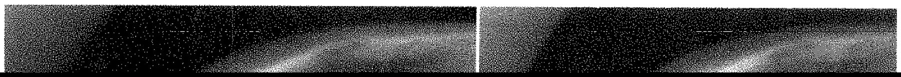

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Atherosclerosis is not commonly found

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such as a meningioma makes this distinc-

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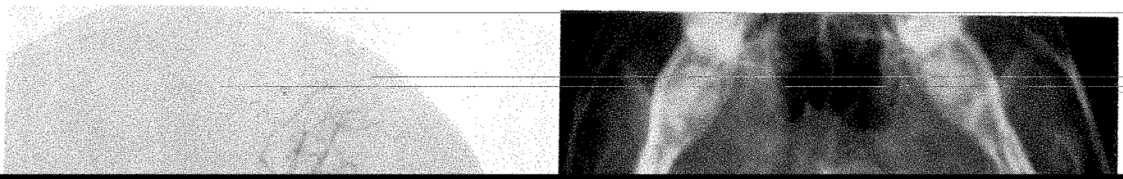

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ANGIOGRAPHY IN POSTERIOR FOSSA TUMORS OF INFANCY AND CHILDHOOD[*]

By SAMUEL M. WOLPERT, M.D.
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IN MANY medical centers the neuroradiologic investigation of posterior fossa tumors in infancy and childhood is confined to a demonstration of the ventricular pathways, usually by pantopaque or air ventriculography. Pneumoencephalography is a study carried out with reluctance by some in the suspected presence of a posterior fossa tumor. Following ventriculography many neurosurgeons will perform a craniectomy either to make a tissue diagnosis, remove part or all of the tumor or to relieve ventricular obstruction.

In our institution, angiographies are always performed as a preliminary investigation prior to air studies. The findings on angiography determine the next procedure to be followed. If ventricular enlargement or tonsillar or transtentorial herniation is present, ventriculography is performed. When neither abnormality is found a careful pneumoencephalography is usually carried out next.

This paper will present a series of selected cases in which the diagnostic yield of posterior fossa angiography in the pediatric age group is demonstrated.

METHODS

While posterior fossa angiography in adults has become widely accepted as a practical procedure,[22] its relative neglect in childhood and infancy is probably largely due to difficulties in the performance of angiography in this age group. However, a series of techniques is available[1,4,6,13,16] and with experience angiography of the posterior fossa can be successfully carried out in patients as young as the newborn. Initially, right brachial angiography was our procedure of choice. The right brachial approach was selected so that in addition to visualization of the posterior fossa, an assessment of ventricular size could be made by visualization of the subependymal veins. The disadvantage of this procedure is, however, that in the frontal view the anterior cerebral artery and its branches tend to obscure the posterior inferior cerebellar artery. As the posterior inferior cerebellar artery is one of the more important vessels in the elucidation of the cerebellar tumors, the disadvantages of this technique soon became apparent. More recently, transfemoral catheterization of the vertebral artery, as described by Radner,[21] and Cronqvist,[3] and subsequently by Newton and Gooding[19] for the pediatric age group has been adopted. A combination of meperidine, promezathine and chlorpromazine was generally adequate for anesthetic purposes and few cases needed general anesthesia. Usually the left vertebral artery was selected as the vessel was somewhat easier to catheterize than the right. Approximately 4–6 ml. renografin 60 was injected by hand. In all cases, the catheter was subsequently manipulated into either common carotid artery and a further 6–8 ml. renografin injected. The purpose of this second injection was to assess ventricular size.

RESULTS AND DISCUSSION

A. INFERIOR CEREBELLAR TUMORS

Tumors in this group include cerebellar astrocytomas, medulloblastomas and ependymomas. The value of angiography is probably greatest in this group due to the strategic situation of the posterior inferior cerebellar artery. The anatomy of this artery has been described by several authors,[7,8,10,27] and the description that follows relies considerably on the nomenclature used by Huang and Wolf.[10]

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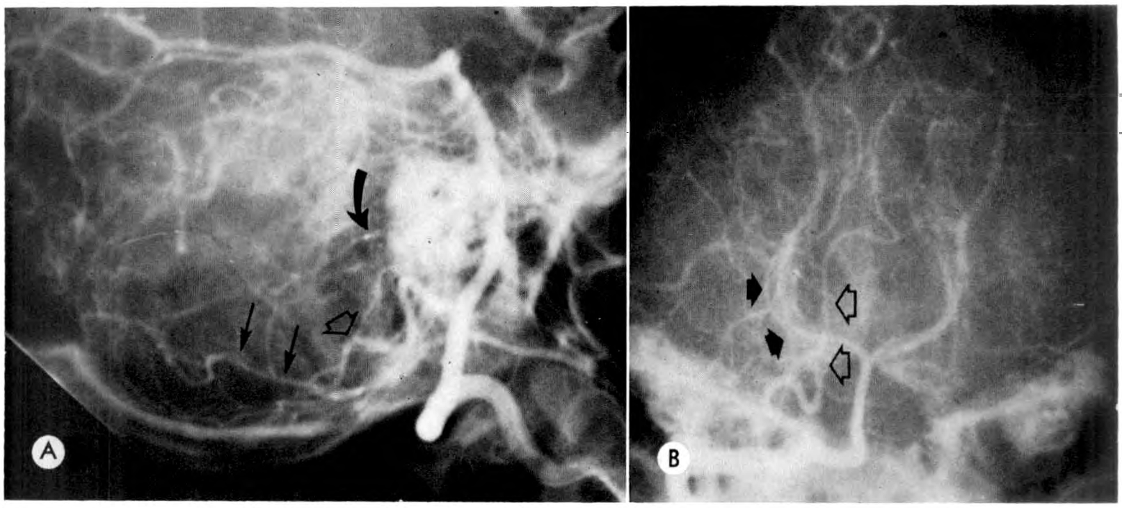


FIG. 1. Male 5 years of age.

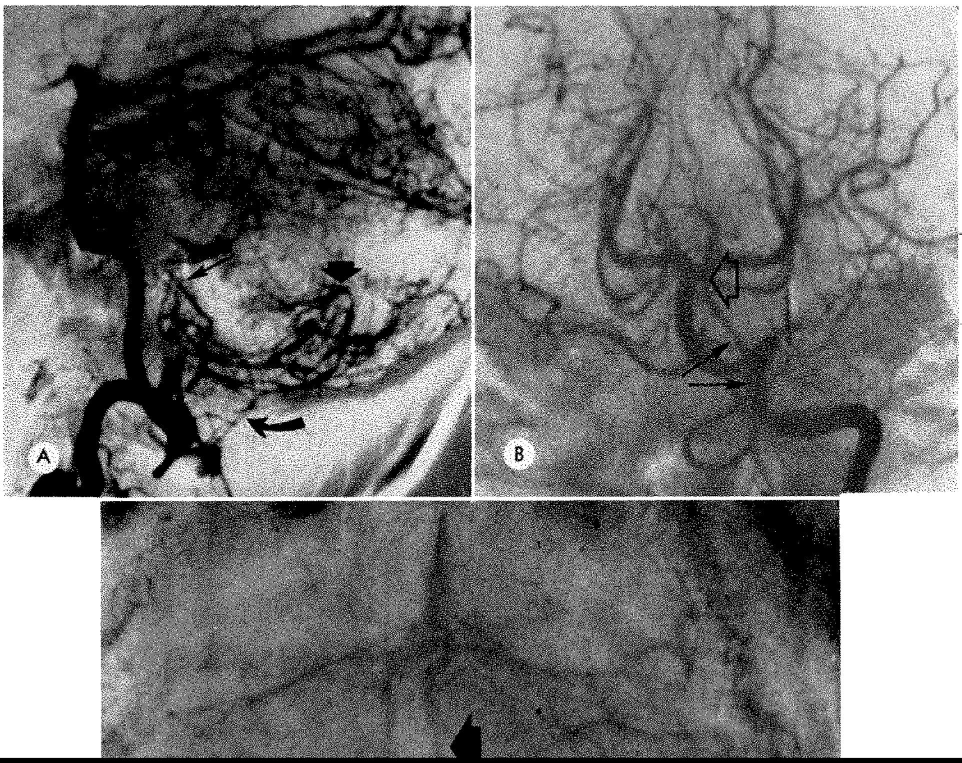
(*A*) Lateral view of the arterial phase of right brachial angiography showing downward displacement of the medial tonsillar (open arrow) and retrotonsillar (closed arrows) branches of the posterior inferior cerebellar artery. A choroid branch (curved arrow) is displaced anteriorly due to the growth of the tumor posteriorly and inferiorly in the region of the fastigium of the fourth ventricle. (*B*) Half axial view of the arterial phase shows lateral displacement of the medial tonsillar and retrotonsillar branches (open arrows). The choroid branch (closed arrows) is displaced markedly laterally.

In 1 patient (Fig. 1, *A* and *B*) the medial tonsillar and superior retrotonsillar segments were displaced and stretched inferiorly and laterally. These changes according to Huang and Wolf[10] suggested a fourth ventricle tumor. At surgery a medulloblastoma originating from the fourth ventricle and separating the tonsils was found. In another patient (Fig. 2, *A–C*), the choroid point was not present, the paratonsillar segments were displaced downwards and laterally with an increase in the distance between the posterior medullary segment and superior retrotonsillar segment. However, the proximal vermial segments were in the midline suggesting either that the mass had not as yet grown to involve the vermis or that the cerebellar hemisphere was infiltrated with tumor. The latter was suggested by displacement of the left inferior vein to the right. At surgery, an ependymoma was found growing out from the fourth ventricle. In a third patient (Fig. 3, *A–C*) the superior retrotonsillar vermial segment and tonsillo-hemispheric segments were draped around a mass lesion which was calcified on routine skull roentgenograms. In the frontal view marked deviation across the midline of the retrotonsillar segments was seen. The precentral cerebellar vein[9] was displaced anteriorly. The inferior vermial vein was displaced laterally. At operation, a cerebellar oligodendroglioma originating from the left tonsil was removed.

In addition to the tumors already mentioned, extra-axial leptomeningeal cysts of the posterior fossa may present as inferior cerebellar hemispheric masses. Their angiographic appearances have been previously described.[28]

B. SUPERIOR CEREBELLAR TUMORS

Tumors in this group include cerebellar astrocytomas and medulloblastomas. The superior cerebellar artery is stretched and elevated.[14] Mani and Newton[17] commented that if the tumor is unilaterally situated the marginal branch is stretched. The venous changes to be expected would include anterior displacement of the precentral cerebellar vein and stretching of the superior vermial vein. When the tumor is large, upwards herniation of the vermis and brain



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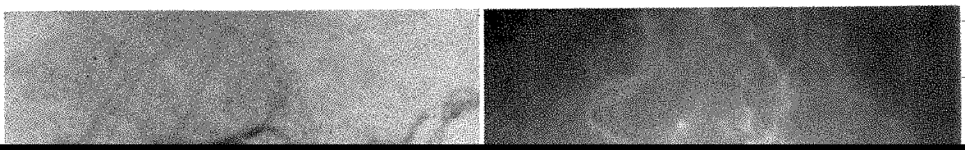

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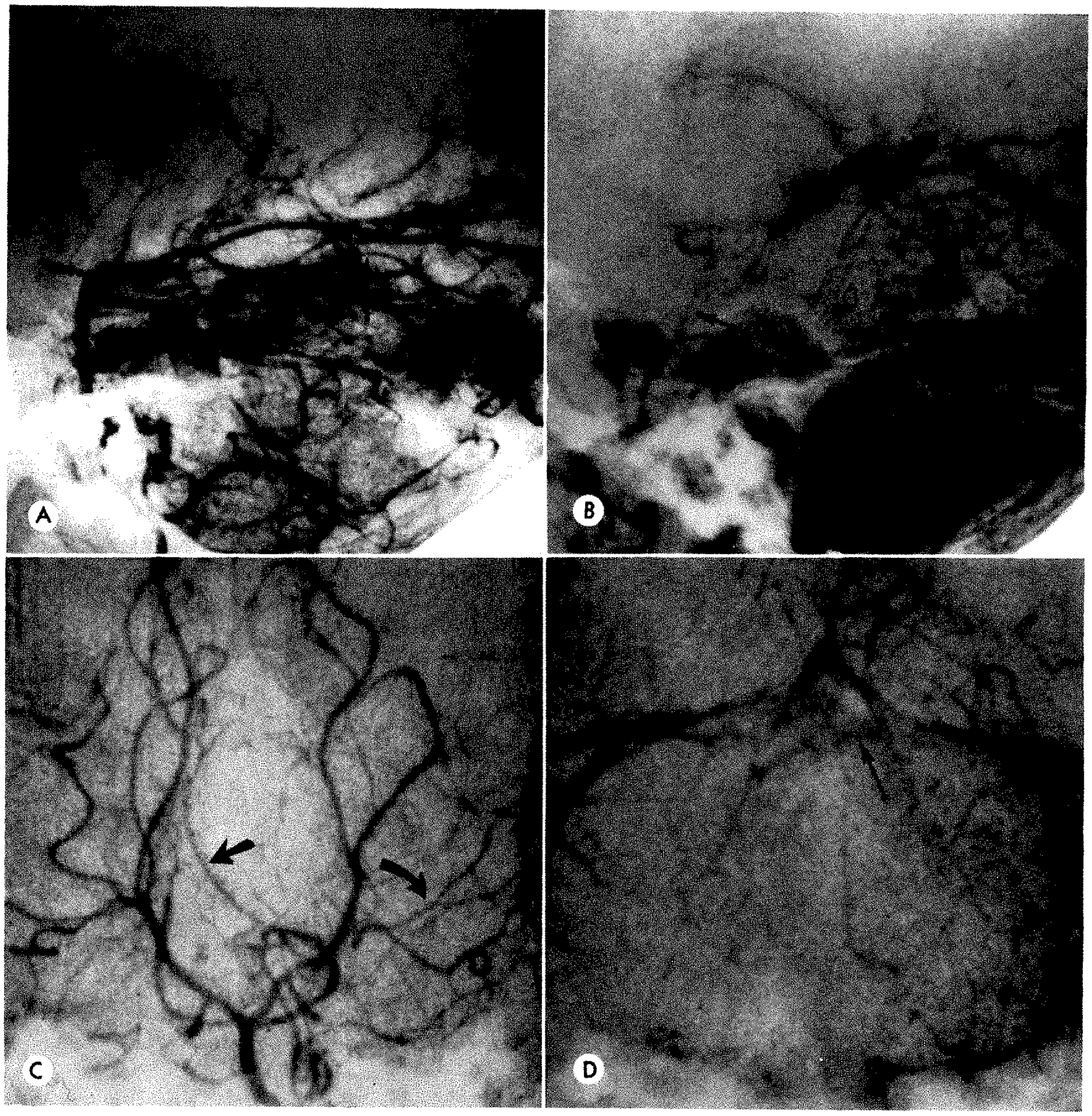
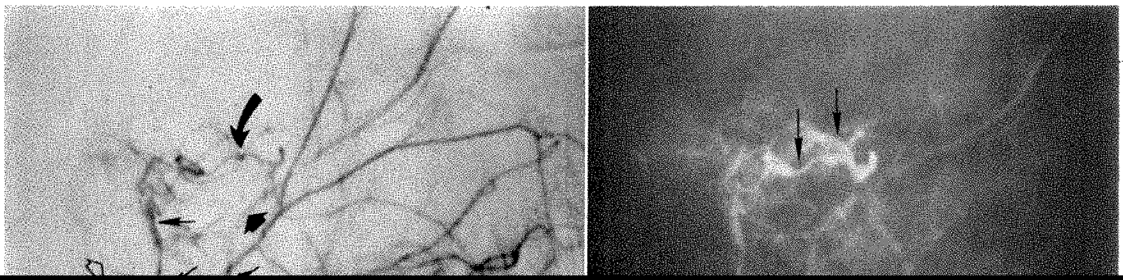


Fig. 4. Female 2 years of age.

(*A*) Lateral view of the arterial phase of left vertebral angiography demonstrates stretching of the superior vermial branch of the superior cerebellar artery (small arrow) and anterior displacement of the basilar artery (large arrow). (*B*) Lateral view of the venous phase shows upward displacement of superior vermial vein (large arrow), anterior displacement of precentral cerebellar vein (open arrow) and anterior displacement of pontomesencephalic vein (small arrow). (*C*) Half-axial view of the arterial phase demonstrates displacement of superior vermial branch of left superior cerebellar artery to right of midline (straight arrow) and lateral displacement and stretching of the marginal branch (curved arrow). (*D*) Half-axial view of the venous phase shows posterior displacement and tilting of posterior mesencephalic vein on the left side.



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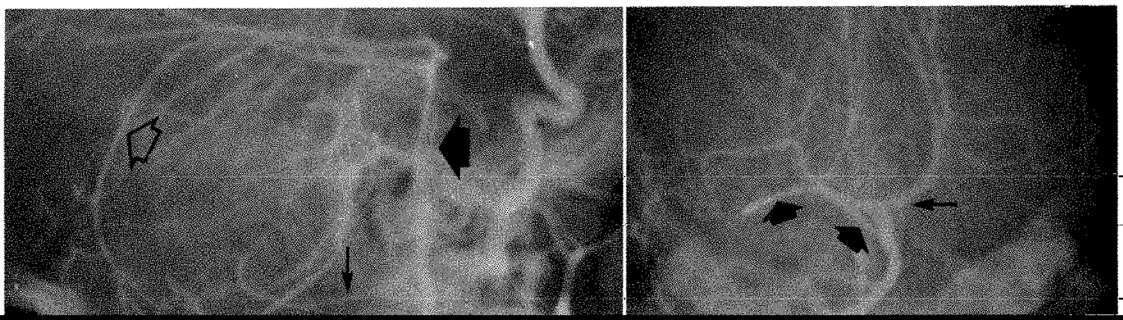

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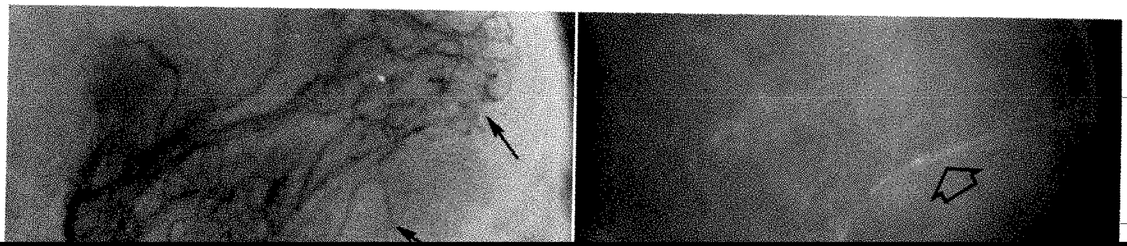

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RADIONUCLIDE EVALUATION OF THE DANDY-WALKER MALFORMATION AND CONGENITAL

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as recorded by brain scintigrams and an additional 10 examples of Dandy-Walker cyst from our teaching files as recorded by angiography. The mean torcular angle was found to be 110° with a standard deviation of 10°. A range from 88° to 117° was documented. A marked difference in the torcular angle between the congenital malformations of the posterior fossa and the normal is illustrated in Figure 1.

Radionuclide ventriculography and cisternography were performed with high specific activity I^{131} human albumin after blocking the thyroid gland with Lugol's solution. Intraventricular injections of 25 μc and subarachnoid injections of 75 μc were followed by multiple scintigrams in

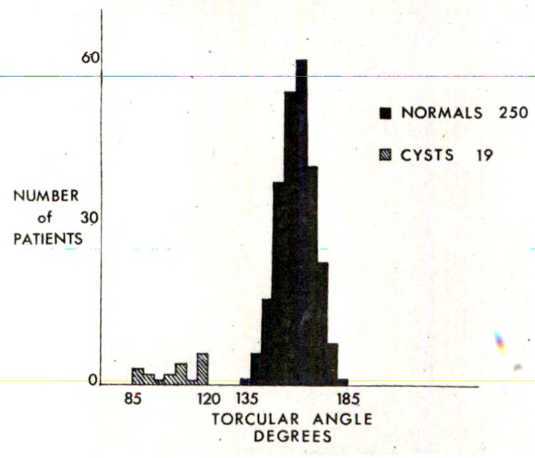

FIG. 1. Comparison of the angle of the transverse sinuses at the torcular (torcular angle) between "normal patients" and patients with cystic mal-

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TABLE II

PATIENT DATA

Patient	Sex	Age	Symptoms	Electroencephalogram	Head Size	Fontanelle	Torcular Angle	Diagnosis
1. T.D.	F	4 da.	0	Not done	Increased	Full	88°	Dandy-Walker; agenesis of corpus callosum
2. R.D.	M	5 mo.	0	Normal	Increased, dolichocephalic shape	Sunken	90°	Dandy-Walker; agenesis of corpus callosum
3. M.A.	M	9 wk.	0	Not done	Increased	Sunken	100°	Dandy-Walker; agenesis of corpus callosum
4. M.O.	M	6 wk.	Seizures	Normal	Increased	Full	110°	Dandy-Walker
5. M.J.	M	2 yr.	Psychomotor retardation ataxia	Dysrhythmia Grade II. Generalized delta Grade I	Increased	Not noted	117°	Dandy-Walker
6. M.V.	F	6 yr.	Psychomotor retardation seizures	Not done	Increased	Closed	116°	Dandy-Walker
7. J.S.	F	3 wk.	Seizure	Normal	Normal	Sunken	110°	Arachnoid cyst
8. B.M.	F	1 yr.	Seizures	Dysrhythmia Grade I	Increased, dolichocephalic shape	Sunken	117°	Arachnoid cyst
9. T.O.	F	8 mo.	Psychomotor retardation	Dysrhythmia Grade II. Generalized	Increased	Sunken	111°	Arachnoid cyst

The transverse sinuses may also be displaced in a cephalic direction (Fig. 10), but this has been an inconstant finding and thus is less reliable. Hypoplastic sinuses may not be visualized. In infants, the small size of the cranial vault may hinder visualization of the sinuses due to insufficient resolution of the recording system.

The transverse sinuses can travel along the posterior occipital wall before migrating anteriorly and thus may appear normal. In addition, a false displacement of the sinuses may be suggested as a result of malposition.

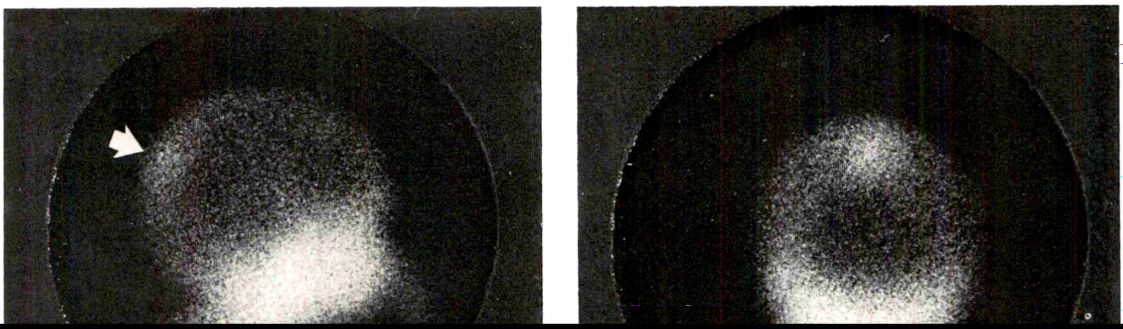
OTHER VIEWS

The anterior, anterior orbit and vertex views were normal in our series.

RADIONUCLIDE VENTRICULOGRAPHY AND CISTERNOGRAPHY

In 2 patients the Dandy-Walker cyst filled readily from a ventricular injection of high specific activity I^{131} human serum albumin (Fig. 7; and 8). The subarachnoid spaces did not fill from the cyst.

In 1 patient a subarachnoid cyst in the posterior fossa was filled from a lumbar in-



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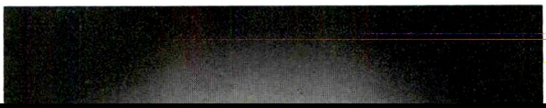

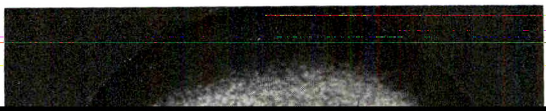

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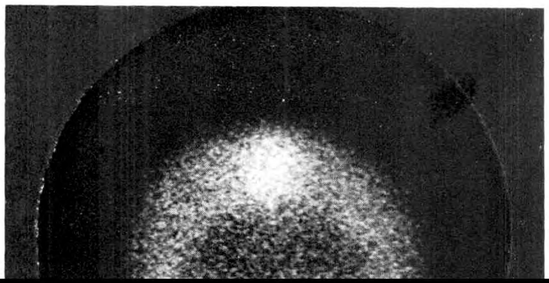

should not visualize following a radionuclide ventricular injection. Such was the case in the 2 patients in this series. However, this may not be an absolute finding, for the Dandy-Walker malformation has been reported with patency of the foramina of Magendie.[1] It has also recently been reported[5] that absorption of I^{131} human serum

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CEREBRAL ANGIOAUTOTOMOGRAPHY*

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into position Q. The 3° angle illustrated has no special significance, but has been arbitrarily chosen to demonstrate the principles involved when using short exposures covering only a fraction of a rotation.

It is seen that the unsharpness of the projected point P, located well away from the midline of the skull, is sufficient to produce considerable blurring. On the other hand, the projections, R′ of point R are considerably sharper, even though they are shown as having been carried through the total angle of 10°. Therefore, it would seem reasonable that points in the lateral aspect of the skull may be adequately blurred during an extremely short exposure encompassing only a part of a rotation. Although exposure times of 3 and 4 seconds are frequently mentioned in the liter-

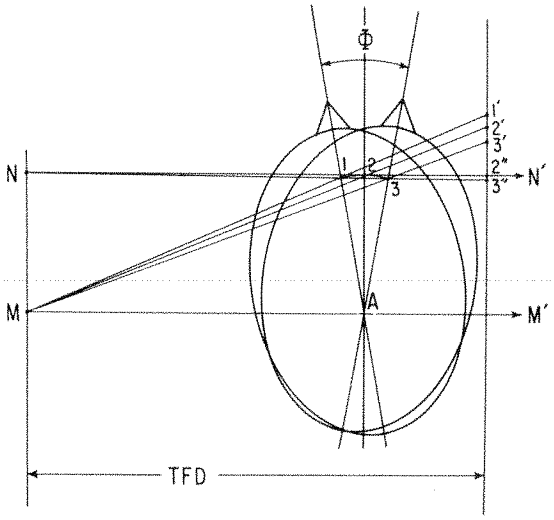

Fig. 10. Rotation of a midline point through angle, Φ. Line M-M′ illustrates the central ray passing through the axis of rotation, A. Line N-N′ shows

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projection on the film plane of a point as

moencephalography using longer exposures.

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supine with the head resting on the occiput and the technique of angioautotomography requires rotating the patient's head by grasping the nose.

This, of necessity, places the axis of rotation far posteriorly, well within the posterior fossa. Alteration of the axis of rotation by flexion or extension is technically more difficult, primarily because of the presence of the needle in the carotid artery. Because of the posterior location of the axis of rotation, the majority of the midline structures of the brain will be more than 4 cm. removed from the axis. The same calculations of New and Webster[8] show that when the midline point under consideration is more than 4 cm. removed from the axis of rotation, unilateral rotation; *i.e.*, asymmetrically from a plane parallel to the film to the side away from the film, will give the least unsharpness. When the rotation is in the reverse direction, from the vertical towards the film, much more unsharpness results. In angioautotomography we are dealing with short exposure times which will, at random, catch the head in one or the other modes of unilateral rotation. This is felt to explain, at least in part, why varying degrees of relative tomographic quality are seen in a

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disease affecting the midline structures of the brain.

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TUMOR BULGE INTO THE SPHENOID SINUS*

A ROENTGEN SIGN OF PARASELLAR MENINGIOMA

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THE patient with a sphenoid ridge or middle fossa meningioma can present as a perplexing diagnostic problem. These tumors are occult in many instances, and it is not unusual to have repeatedly normal roentgenologic studies.[1] Specific plain skull roentgenographic changes suggesting the diagnosis are reported to be found in only about 50 per cent of the cases.[6,12,17]

In recent years, 5 patients have been admitted to this hospital with only equivocal radiodensity in the region of the sphenoid wings evident on plain skull roentgenograms. Tomograms were then obtained through the sphenoid structures and a smooth tumor mass was found to be bulging into the air shadow of the sphenoid sinus. A meningioma was surgically verified in each case.

The details of 3 of these cases are presented in this report to illustrate the pertinent roentgenographic findings.

REPORT OF CASES

CASE I. R.H., a 55 year old white female with a 1 year history of proptosis and decreasing vision of the left eye, was admitted to this hospital on June 25, 1966. Funduscopic examination revealed optic atrophy on the left. The findings on skull roentgenograms were limited to radiodensity of the left sphenoid wings of questionable significance (Fig. 1*A*). Tomography (Fig. 1*B*) clearly demonstrated a bulge into the left side of the sphenoid sinus. Subsequent brain scanning and left carotid arteriography showed a huge tumor mass in the middle fossa. The patient underwent a left frontotemporal craniotomy with resection of a large meningioma in the middle fossa. The dural attachment extended to the medial two-thirds of the lesser wing of the sphenoid and the medial half of the middle fossa. The tumor also extended backward over the tentorium into the posterior fossa as well as forward into the orbit and medially into the cavernous sinus.

CASE II. M.R.P., a 37 year old white female, was originally admitted to this hospital on March 18, 1965 for evaluation of headache in the orbital region of 6 months' duration with visual difficulty and diplopia. In addition, the patient had a long history of migraine headaches. She was said to have sustained head trauma with a questionable skull fracture at the age of 15 years, left mastoiditis at the age of 16 years, and meningitis at the age of 27 years. Examination revealed decreased visual acuity of the left eye, slight ptosis, weakness in the distribution of the left oculomotor nerve, and anisocoria with the left pupil larger than the right. Skull roentgenograms and left carotid arteriography were normal and the patient was discharged with the diagnosis of ophthalmoplegic migraine.

She was re-admitted on October 2, 1967 with complaints of left retro-orbital pain, numbness of the left side of her face, and visual impairment. Examination again showed the anisocoria and sensory loss of the fifth cranial nerve on the left. Electroencephalogram and brain scan were normal.

Skull roentgenograms revealed a small left superior orbital fissure, together with relative enlargement and questionable sclerosis of the left anterior clinoid process and lesser sphenoid wing (Fig. 2*A*). Subsequent tomograms showed the sclerosis as noted above together with a smooth bulge into the sphenoid sinus on the left (Fig. 2*B*). Left carotid arteriography demonstrated only narrowing of the internal carotid artery in its cavernous portion and pneumoencephalography revealed a slight upward displacement of the left temporal horn. Left frontal craniotomy was then carried out with partial resection of a meningioma extending over the left greater and lesser wings of the sphenoid and extending down over the roof of the left cavernous sinus.

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Case iii. H.J.W., a 22 year old Negro male, was admitted to this hospital on December 12, 1966 for recurrent proptosis of the left eye. A left orbital exploration had been carried out in 1964 following a 3 year history of blindness of the left eye with proptosis and optic atrophy. A meningioma of the apex of the orbit was found and partially removed. Skull roentgenograms

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Meningiomas of the parasellar region can

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tumor bulge in the sphenoid sinus, the diagnosis is more uncertain than when hyperostosis is present. This combination of roentgen changes is demonstrated in Figure 4, *A* and *B*, secondary to an extradural hemangiopericytoma at the base of the middle fossa which had extended to the cavernous sinus.

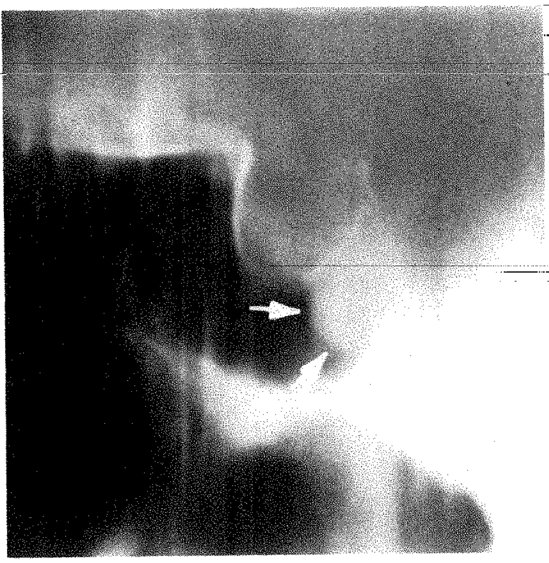
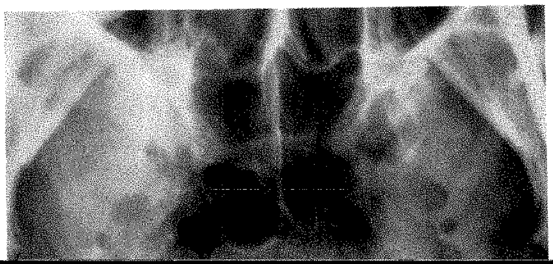


Fig. 5. Chromophobe adenoma. Lateral tomogram of sella turcica demonstrating tumor bulge into the sphenoid sinus (arrows). Transphenoidal removal.

We have not seen both hyperostosis and a smooth tumor bulge into the sphenoid sinus in tumors other than meningioma. Tumor bulge into the sphenoid sinus can also be found in pituitary adenoma[9] (Fig. 5). Our cases of malignancy involving the sphenoid sinus have demonstrated only irregular contours of the involved sphenoid sinus wall at tomography. This has also been the experience of others.[8] Hyperostosis of the skull base has been found in osteoblastic metastasis and can simulate changes seen in the *en-plaque* meningiomas in this area.[5,14] However, a smooth bulge into the sphenoid sinus has not been described with cancer. A solitary osteoblastic metastasis in



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BRAIN TUMORS IN CHILDREN WITH NORMAL SKULL ROENTGENOGRAMS*

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THE skull roentgenograms of 50 consecutive children with proven brain tumors were reviewed because the reported incidence of bony calvarial abnormalities of 70–80 per cent in retrospective studies seemed high.[2–4]

The purpose of this study is to emphasize the need for further neuroradiographic procedures despite normal skull roentgenograms if the signs and symptoms suggest a neoplasm. The possible reasons for the difference between the findings in this series and those found in the literature are discussed.

RESULTS

The skull roentgenograms of each patient were interpreted independently by 2 or more of the authors. Twenty-three of the 50 children with proven brain tumors had normal skull roentgenograms. The mean age of the patients with normal skull roentgenograms was 9 years, whereas those children with abnormal skull roentgenograms had a mean age of 6 years (Fig. 1). The difference in age distribution is statistically significant ($p = 0.01$).

The duration of signs and symptoms, before the diagnosis of brain tumor was made, was 5.7 months for the children with normal skulls and 6.4 months for those children with abnormal skulls. This slight difference is not significant. Patients in both groups showed signs and symptoms of headache, vomiting, eye signs and gait abnormalities.

Of the 27 children with abnormal skull findings (Table 1) 20 had suture diastasis. Eleven of these 20 patients had no other changes in the skull. Five children in addition to the abnormal sutures had enlarged calvaria. Of the 4 remaining patients with wide sutures 1 had a demineralized sella, 1 had calcification in the occipital lobe and a large occipital emissary vein and 2 had suprasellar calcifications with expanded sellae.

The remaining 7 patients with abnormal skull roentgenograms had normal sutures. One child had a calcification of the left posterior temporal region with thinning of the bone over this area. Two patients had enlarged sellae secondary to intrasellar tumors. One patient had a tumor anterior to the third ventricle extending to the suprasellar region causing destruction of the floor of the sella and of the posterior clinoids. Cranial enlargement with prominent fontanelles was an isolated finding in 3 infants 18 months or younger.

No correlation was noted between the incidence of abnormal skull roentgenograms

Table I

CHILDREN WITH BRAIN TUMORS AND ABNORMAL SKULL FINDINGS

Sutural Diastases			20
No other changes		11	
Enlarged calvaria		5	
Large fontanelle	2		
Suprasellar calcifications		2	
Calcified occipital tumor and large emissary vein		1	
Demineralized sella		1	
Abnormal Skulls, Sutures Normal			7
Enlarged calvaria and fontanelle		3	
Intrasellar expansion		2	
Parasellar tumor with destruction of floor of sella		1	
Calcification in left posterior temporal region with thinning of overlying bone		1	

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* Presented at the Thirteenth Annual Meeting of the Society for Pediatric Radiology, Miami Beach, Florida, September 28, 1970.
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mon change in the skull of children with a brain tumor. Calcifications and alterations in the sella are also reliable findings. Enlargement of the bony channel carrying the occipital emissary vein over 2 mm. in size is occasionally seen as a result of increased intracranial pressure.

The incidence of 54 per cent of abnormal skull findings in the children presented in this study is lower than reported by others.[2–4] Prominent convolutional markings of the inner table and large vascular channels are often seen in normal children. Therefore, these findings alone should not be considered specific signs of increased intracranial pressure.[5]

The lower incidence of abnormal skull findings in children with brain tumors in this series may not only be due to more specific criteria but may also be due to earlier diagnosis. It is important that a neuroradiologic work-up be done in spite of normal skull roentgenograms, if there are any clinical signs or symptoms to suspect a brain tumor.

Table II B

TYPE OF TUMOR

Abnormal Skulls			27
Supratentorial			10
Cerebral		5	
Astrocytoma	4		
Ependymoma	1		
Sellar and Parasellar		5	
Pinealoma	1		
Craniopharyngioma	1		
Chromophobe adenoma	2		
Astrocytoma	1		
Infratentorial			17
Cerebellar		14	
Medulloblastoma	6		
Astrocytoma	7		
Ependymoma	1		
Brain Stem		3	
Glioma	2		
Astrocytoma	1		

CONCLUSION AND SUMMARY

1. Forty-six per cent of children with brain tumors in this series had normal skull roentgenograms.
2. Convolutional markings or increased vascularity in the calvaria are unreliable indicators of brain tumor.
3. No correlation was noted between the incidence of abnormal skull roentgenograms and the location or the histology of the brain tumor.
4. Neuroradiologic work-up should be done in all children with suspected brain tumor in spite of normal skull roentgenograms.

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ANGIOGRAPHY IN ANGIOMAS OF THE FACE*

By M. LEA THOMAS and M. R. ANDRESS

LONDON, ENGLAND

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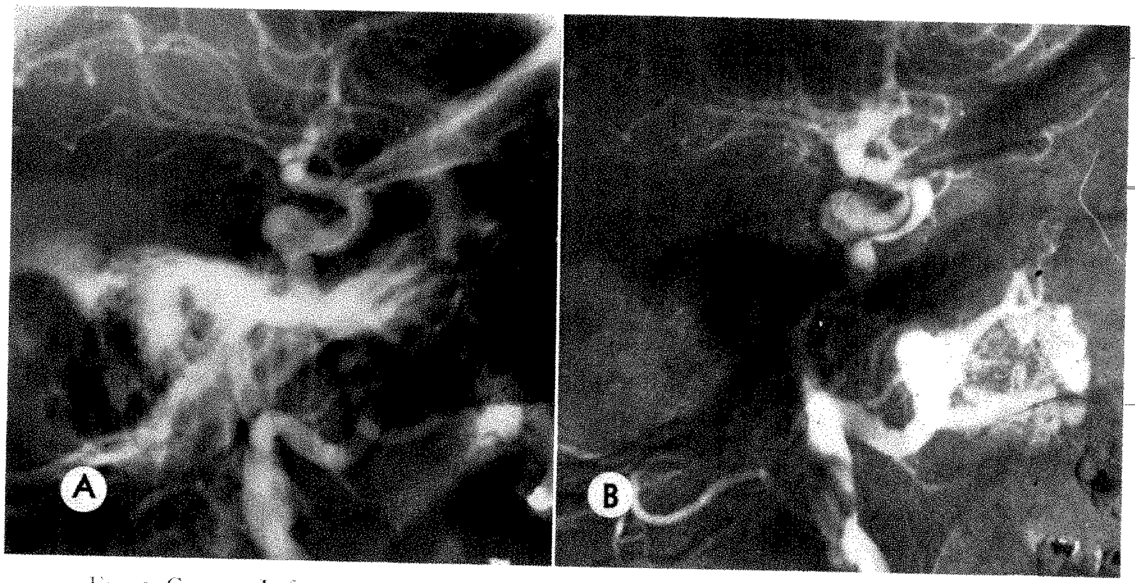

FIG. 2. Case III. Left common carotid arteriogram. (*A*) Conventional lateral angiogram. (*B*) Subtraction film. Note enlarged maxillary artery and angioma in infratemporal fossa.

in size and this had raised the possibility that the lesion might be a malignant growth.

Left common carotid arteriography showed a vascular mass 4 cm. in diameter in the infratemporal fossa, supplied by a greatly enlarged internal maxillary artery (Fig. 2, *A* and *B*). There was early venous drainage into the internal jugular vein. There was no pooling of contrast material and the appearances were those of an arteriovenous malformation. No supply was demonstrated from the right carotid arterial system and there was no evidence of intracranial extension. As the lesion was causing little disability, operation was not considered necessary and the patient was reassured about the benign nature of the lesion.

CASE IV. A 60 year old woman had a painful swelling of the tip of her nose for 3 years and had had one large nose bleed. On examination

the months since the operation the patient's progress has been satisfactory.

CASE V. A 41 year old doctor had an angioma of the face, mainly on the right side, which had been present since birth. The right external carotid artery had been ligated at the age of 29 years. Since then he had required several further operations. These were not preceded by preoperative angiography. The present admission was because of severe exsanguinating epistaxes.

On examination there was a recurrent angioma mainly in the right nasal orifice (the nose having been removed). There were pulsating feeding vessels in the right lip and maxillary region.

Right common carotid arteriography showed the previous ligation of the external carotid artery.

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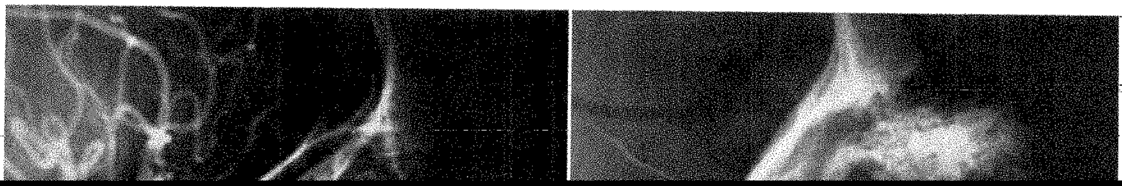

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operative progress was satisfactory but the child could not be followed up as he came from abroad.

DISCUSSION

[redacted]

extent and relationship of the angioma we believe that 3 projections are required: lateral; 30° mento-occipital; and submento-vertical, coned for the facial region.

Due to the complex structure of the facial

In the 1 patient in whom malignancy was considered, reassurance proved invaluable in management.

SUMMARY

Seven patients with the uncommon condition of angioma of the face have been investigated.

venous fistulas and congenital aneurysms. *J. Cardiovasc. Surg.*, 1967, *8*, 209–215.
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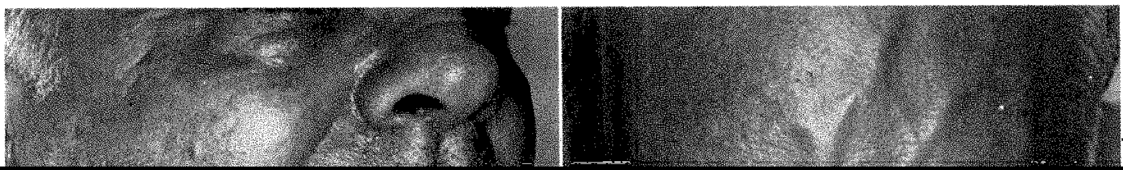

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POSITIVE CONTRAST ORBITOGRAPHY*

By WILLIAM N. HANAFEE, M.D.,† ROBERT S. HEPLER, M.D.,‡ *and*
CARL G. COIN, M.D.§
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DILUTE water soluble contrast agents form a more homogeneous distribution and are more readily absorbed than air in positive contrast orbitography. Image intensification fluoroscopic control of retrobulbar injections results in improved safety of the procedure and virtually eliminates faulty injections. Excessive volumes of contrast material can be avoided. Any localized areas of nonfilling can be confirmed by directing the needle tip to these areas under fluoroscopic control.

HISTORIC REVIEW

Contrast orbitography began with retrobulbar injections of air by Staunig and Herrenschwand in 1927.[10] The injected air is slowly absorbed and provides a low level of contrast. Tomography in association with pneumo-orbitography improves the level of contrast, but leaves considerable uncertainty whether an area of nonfilling is due to tumor or merely the fact that air did not dissect into that particular fascial plane.

Oily opaque media were introduced in 1927; however, they quickly fell into disrepute because of their tendency to form pools and also because of their lack of resorption.[8,9]

The initial experience of Katz and Ledoux in 1935[3] using water soluble contrast media to determine length of the globe proved the relative safety of the procedure. In 1953 Pettinati[7] injected water soluble media to localize foreign bodies.

The procedure began to gain wider acceptance following the reports of Cowie and Groves[1] and Lombardi.[4] They made carefully planned injections to demonstrate specific anatomic fascial planes. These investigators established a minimum useful concentration of soluble radiopaque contrast agents in the range of 17 per cent and 21 per cent, respectively.

ANATOMY

The major usefulness of the technique involves investigations behind the equator of the globe. Two major fascial planes and one muscular fibroelastic envelope are encountered in this region (Fig. 1, *A–C*). The outer one is the "periorbit," which is in reality, the periosteum of the bones forming the orbital cavity and continues intracranially as the dura plane. The inner is Tenon's capsule, or fascia bulbi, which encloses the globe. It extends anteriorly to the limbus of the cornea and is attached posteriorly to the sheath of the optic nerve. An inner layer of this sheath is loosely attached to the sclera of the globe to allow free movement and becomes deficient at the point of emergence of the optic nerve from the globe.

The intermuscular membrane together with the extraocular muscles enclose a space surrounding the optic nerve within the orbit. This space, because of its surgical importance, is called the "central surgical space" and contains the optic nerve, fat, the ophthalmic artery, the superior ophthalmic vein and small branch vessels and nerves.

The amount of fat contained in the central surgical space will vary considerably depending upon the state of nutrition of the patient. Patient hydration or localized

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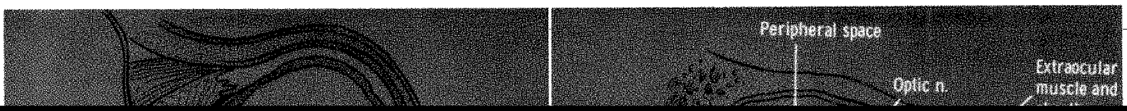

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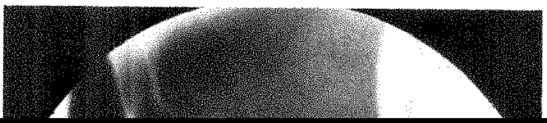

the capability of tomography in either projection.

Under sterile conditions local anesthetic

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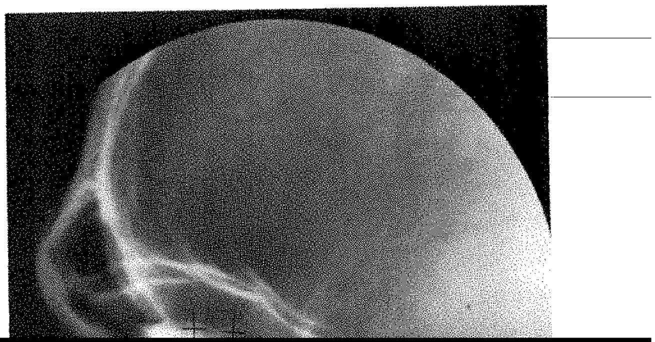
the central surgical space is well known to the ophthalmologist, since this is the standard method of administering anesthesia for surgery on the globe. For the roentgen examination greater care must be exerted to be assured that the contrast material is truly within the central surgical space. The roentgenologic appearance of the cone-shaped central surgical space, as confined by the muscle cone, presents little



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UNUSUAL PENETRATING INJURY OF THE ORBIT*

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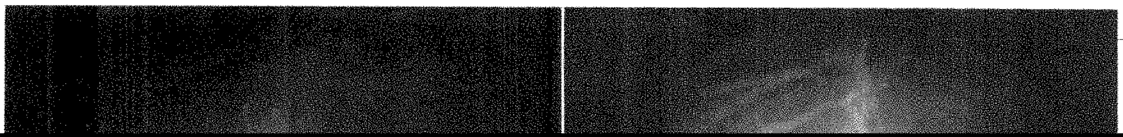

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the surgeon gently eased the knife blade out through the orbit. No hemorrhage or other difficulty occurred.

Culture of the knife blade revealed very few *Staphylococcus aureus*.

Clinical course. The postoperative course was uneventful (Fig. 2*C*). His wounds healed primarily and he was discharged in good condition

damage to the optic nerve and partial damage to the III, IV and VI nerves on the right side.

SUMMARY

A case of unusual penetrating injury of the orbit due to a knife blade is reported.

[redacted]

THE BONY BRIDGE IN OTOROENTGEN DIAGNOSIS*

By LEWIS E. ETTER, M.D.†
PITTSBURGH, PENNSYLVANIA

ROENTGEN examination of *the mastoids* has come to be an almost meaningless term for a roentgen examination of

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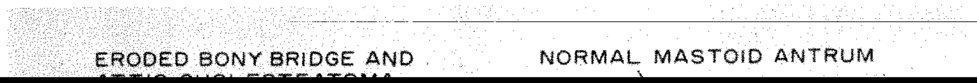
ERODED BONY BRIDGE AND
NORMAL MASTOID ANTRUM

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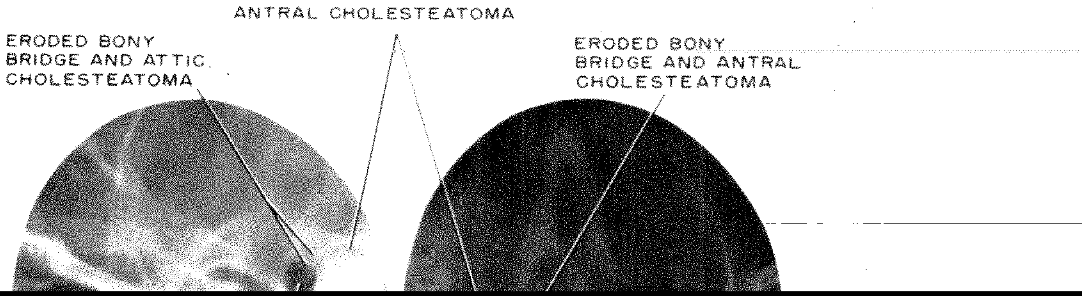
ANTRAL CHOLESTEATOMA
ERODED BONY BRIDGE AND ATTIC CHOLESTEATOMA
ERODED BONY BRIDGE AND ANTRAL CHOLESTEATOMA

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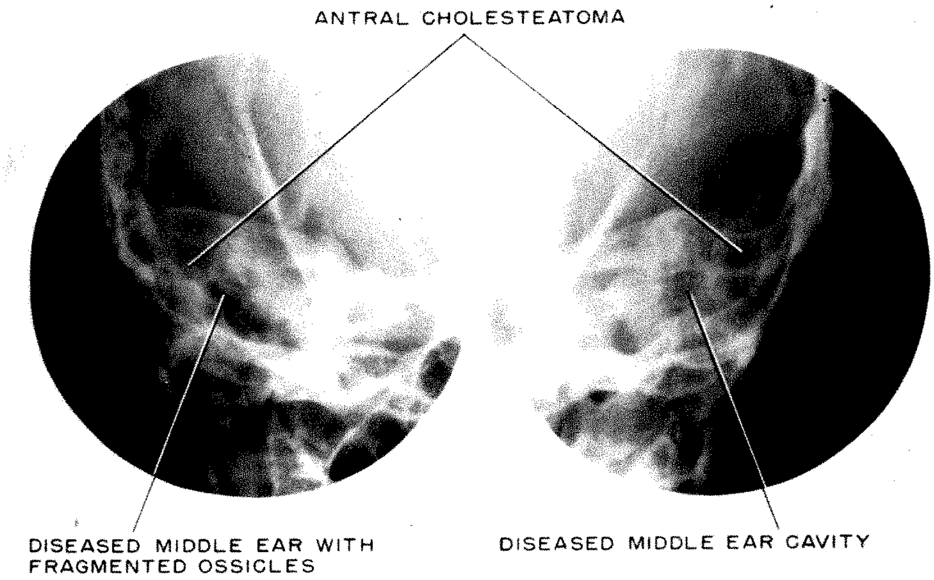
ANTRAL CHOLESTEATOMA
DISEASED MIDDLE EAR WITH FRAGMENTED OSSICLES
DISEASED MIDDLE EAR CAVITY

Fig. 9. The Stenvers projection confirms the changes previously seen, indicating diseased middle ears bilaterally, with fragmented ossicles and bilateral antral cholesteatoma, although, as mentioned in the history of this patient, the left side is presently inspissated.

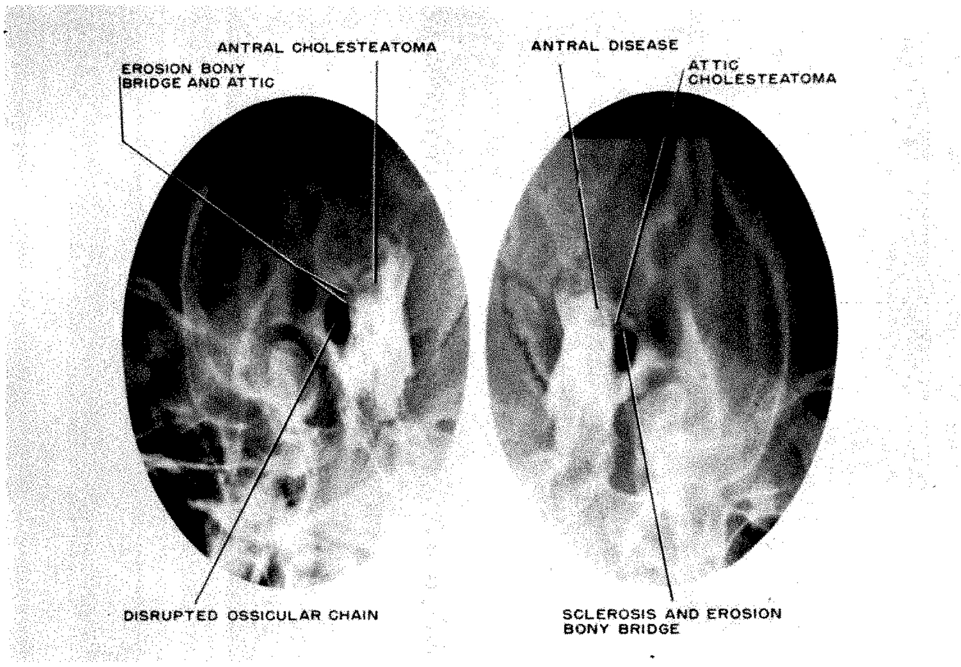
ANTRAL CHOLESTEATOMA
EROSION BONY BRIDGE AND ATTIC
ANTRAL DISEASE
ATTIC CHOLESTEATOMA
DISRUPTED OSSICULAR CHAIN
SCLEROSIS AND EROSION BONY BRIDGE

Fig. 1c. The changes shown in the previous projections are confirmed in this view where there is seen to be disruption of the right ossicular chain with erosion of the bony bridge communicating with an antral cholesteatoma. Similar changes are seen on the left side but there was no drainage at the time of this examination in the Mayer projection.

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ANTRAL CHOLESTEATOMA

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◆

ABNORMAL MAXILLARY ANTRUM IN SUPERIOR ALVEOLAR RIDGE TUMORS AND GRANULOMAS*

By HARVEY I. WILNER, M.D., *and* MEHDI MAHMOODZADEGAN, M.D.
DETROIT, MICHIGAN

THE alveolar arch and bony palate are rarely involved by neoplasms arising either in these structures or in the sur-

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roof of the mouth and also the floor of the maxillary sinuses.[1] The term superior alveolar ridge includes the bony palate, alveolar

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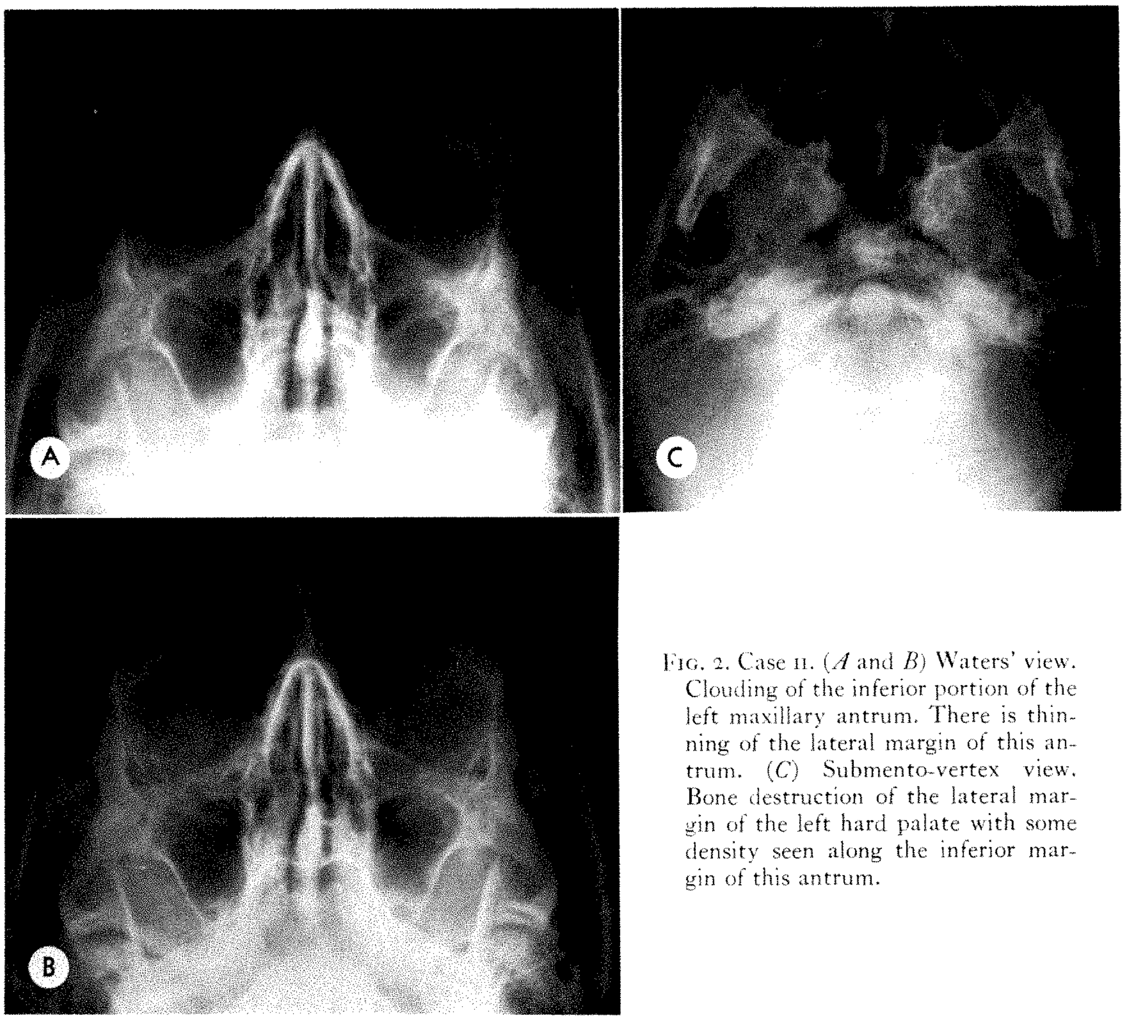


Fig. 2. Case ii. (*A* and *B*) Waters' view. Clouding of the inferior portion of the left maxillary antrum. There is thinning of the lateral margin of this antrum. (*C*) Submento-vertex view. Bone destruction of the lateral margin of the left hard palate with some density seen along the inferior margin of this antrum.

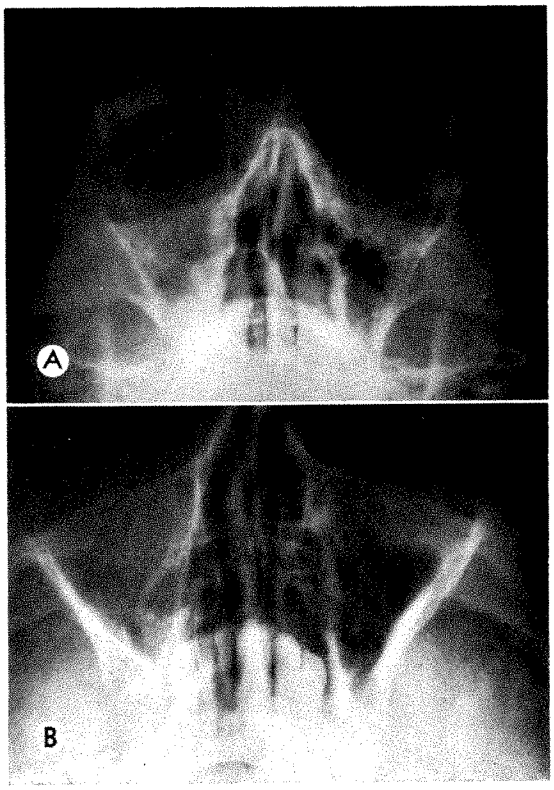


Fig. 3. Case iii. (*A* and *B*) There is thinning of the outer cortex of the lower lateral aspect of the right antrum associated with opacification of this antrum. This is particularly well noted in *B*. The clouding of the right ethmoidal and frontal sinuses is possibly secondary to inflammation related to the tumor.

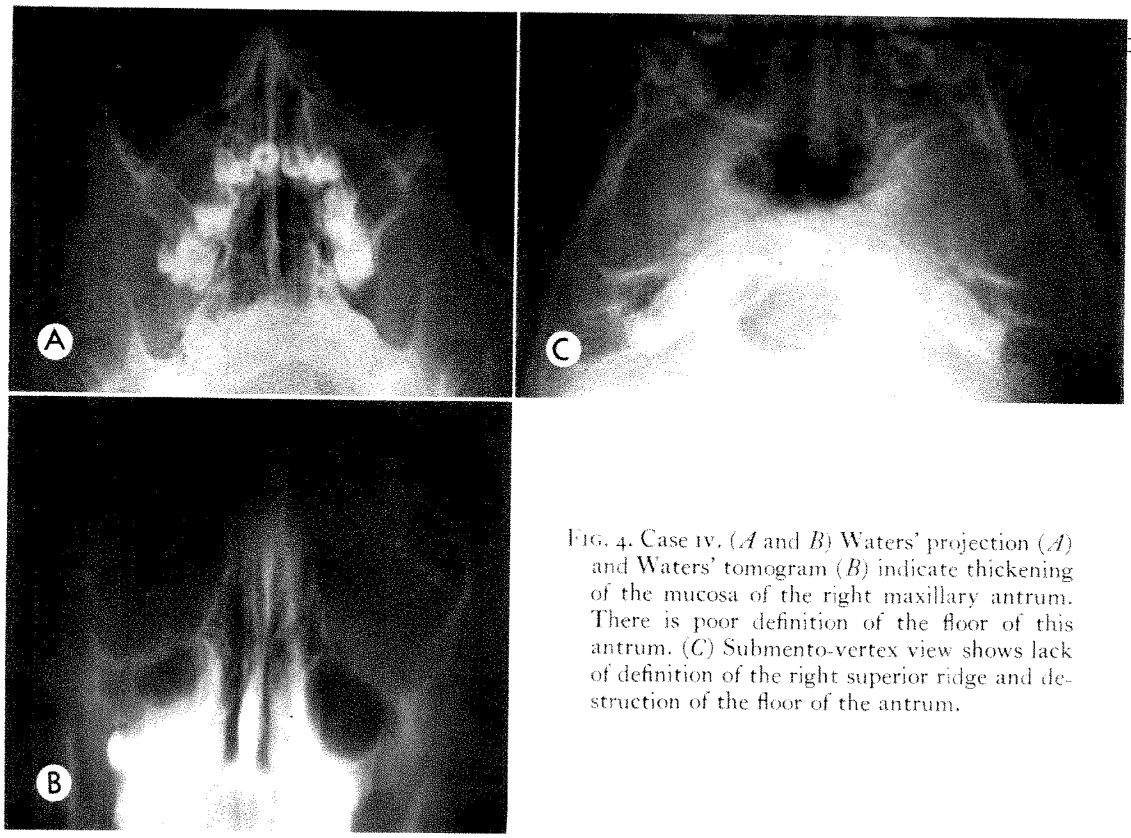


FIG. 4. Case IV. (*A* and *B*) Waters' projection (*A*) and Waters' tomogram (*B*) indicate thickening of the mucosa of the right maxillary antrum. There is poor definition of the floor of this antrum. (*C*) Submento-vertex view shows lack of definition of the right superior ridge and destruction of the floor of the antrum.

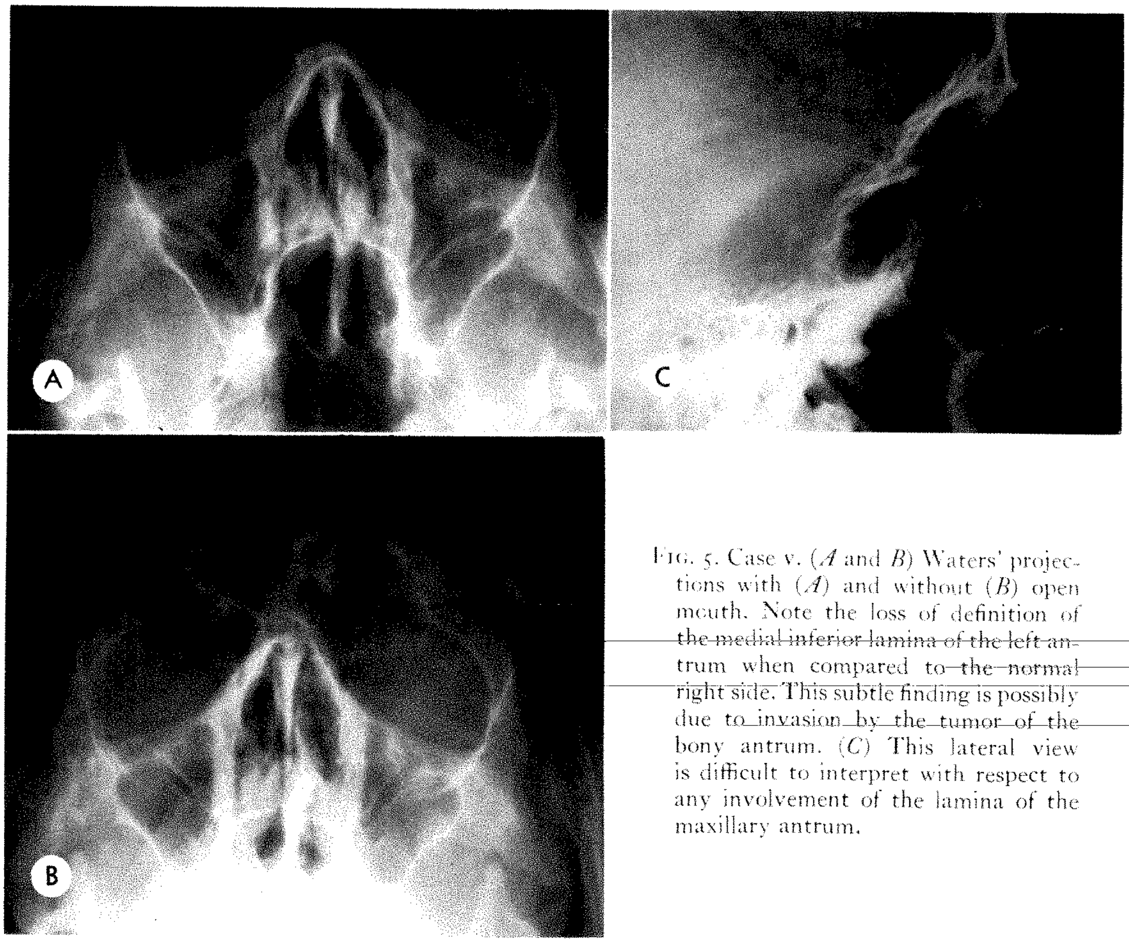
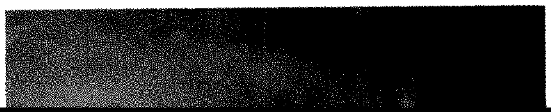


FIG. 5. Case V. (*A* and *B*) Waters' projections with (*A*) and without (*B*) open mouth. Note the loss of definition of the medial inferior lamina of the left antrum when compared to the normal right side. This subtle finding is possibly due to invasion by the tumor of the bony antrum. (*C*) This lateral view is difficult to interpret with respect to any involvement of the lamina of the maxillary antrum.



SUMMARY

Six cases of tumors and 1 case of giant cell reparative granuloma originating from

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ROENTGENOGRAPHIC FINDINGS OF MALIGNANT EXTERNAL OTITIS*

By B. H. KIM, M.D.
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THE entity "malignant external otitis" was first described by Chandler.[1] This disease usually occurs in elderly patients with long-standing and uncontrolled diabetes mellitus. The process follows a malignant course as it progresses rapidly to involve the middle ear, mastoid and petrous pyramid. Once the process has extended beyond the limit of the external auditory canal, it becomes difficult to control it with any kind of treatment.

Since no description of the roentgenographic findings is found in the literature, cases of malignant external otitis recently seen in our institution are reported. All 5 cases underwent conventional and tomographic examinations of the temporal bones.

REPORT OF CASES

CASE I. K.R., 73 year old male. This patient with a long-standing history of diabetes mellitus and congestive heart failure underwent right aural polypectomy and mastoidectomy in another hospital for chronic drainage of the right ear. At the time of admission, the diabetes mellitus was under control with medication, but there was persistent drainage from the right ear. A radical mastoidectomy was then performed. At surgery extensive osteitis of the anterior and inferior walls of the external canal and middle ear was found. A cholesteatoma filled the entire middle ear and external auditory canal. In addition, there was evidence of infection involving the perilabyrinthine cells of the petrous apex. After surgery, the drainage from the right ear persisted, but was not as profuse. The patient died at the convalescent home shortly after discharge. The cause of death was heart failure.

Roentgenographic findings. A tomographic study of the temporal bone was done a few days prior to surgery. No abnormality was detected in the left ear. The findings in the right ear were: extensive destruction of the anterior and inferior wall of the external auditory canal and middle ear cavity; partial destruction of the posterior wall of the external canal; and diffuse clouding of the mastoid air cells. The lateral wall of the attic was intact, but a soft tissue mass obliterated the tympanic cavity. The tegmen was thinned out, but not dehiscent. The facial nerve canal was intact. The lesion extended medially along the inferior aspect of the petrous pyramid, which appeared undermined, to the jugular fossa (Fig. 1, *A* and *B*).

CASE II. M.W., 65 year old female. This patient, with a 20 year history of diabetes mellitus, was first seen in our clinic complaining of severe pain in the left ear and eye of recent onset. Chronic inflammatory changes were noticed in her left ear and a polypoid lesion of the external auditory canal was biopsied. The pathologic examination confirmed chronic inflammatory changes. *Pseudomonas aeruginosa* and *beta hemolytic streptococci* (not Group A) were cultured from the left ear canal. Three months after her first visit, she underwent left radical mastoidectomy. About 2 months later, the patient developed signs of 6th nerve palsy and partial 5th nerve paralysis with decreased corneal reflex. Her condition deteriorated rapidly and she expired 3 months after surgery.

The autopsy findings were acute bronchopneumonia and pulmonary edema, tricuspid and mitral stenosis with multiple kidney infarction and nephrosclerosis, and left petrositis with extensive extradural abscess (Gradenigo's syndrome and mild focal meningitis).

Roentgenographic findings. The roentgenographic examination was performed 1 week prior to surgery. No abnormality was detected in the right ear. The left ear demonstrated destruction of the anterior and inferior wall of the external canal with anterior displacement of the mandibular condyle. Bone destruction and cloudiness were also recognized in the mastoid air cells, located around the external meatus and above the temporomandibular

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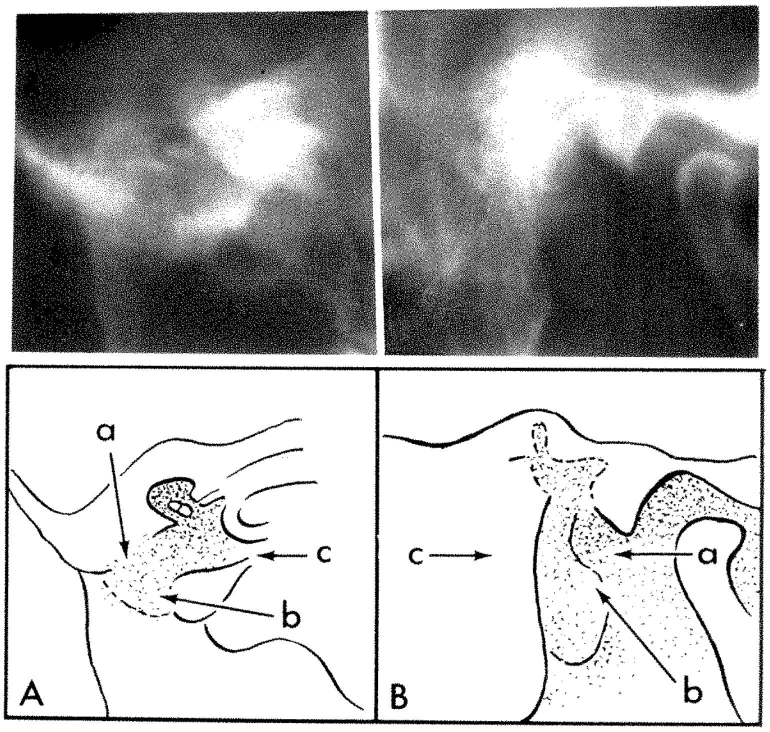


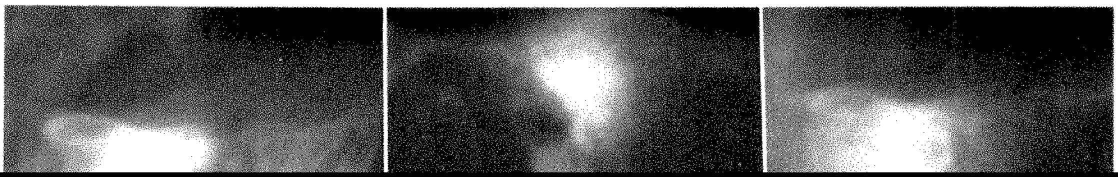
Fig. 1. Case i. (*A*) Frontal section of the tomographic examination reveals destruction of the superior wall (a), inferior wall (b) of the external auditory canal, and blurring of the superolateral wall of the jugular fossa (c). (*B*) Lateral section shows destruction of the anterior wall (a) and the posterior wall (b) of the external canal, and diffuse cloudiness of the mastoid air cells (c).

joint. The middle ear cavity was cloudy, the malleus completely destroyed and the lateral wall of the attic eroded, especially anteriorly. In addition, the tegmen and adjacent petrous ridge appeared ill defined. The first portion of the facial nerve canal was not well visualized and involvement of this area was questioned. The jugular fossa and inner ear structure showed no evidence of destruction. A follow-up examination was done 2 months after operation. The tomograms revealed a surgical defect in the left mastoid and middle ear cavity. However, destruction of the inferior aspect of the petrous pyramid had developed in the meantime. The medial wall of the jugular fossa was partially destroyed (Fig. 2, *A–C*).

Case iii. M.S., 69 year old female. This patient, with long-standing diabetes mellitus and hypertension, was admitted to our institution

[redacted]

nerve paralysis at the time of admission, but signs of left facial weakness developed just prior to surgery. Several cultures revealed *Pseudomonas aeruginosa* from both external auditory canals and occasionally enterococci and *Staphylococcus aureus*. Surgery on the left ear was performed first. As the granulation tissue was removed the cartilage and bony wall of the external canal were found to be intact. The facial canal was intact but an abscess cavity was found just below the tympanic bone in the region of the stylomastoid foramen. The tympanic membrane was normal. The right ear was explored a week later and osteochondritis involving the inferior canal wall was found. The tympanic membrane was intact. The postoperative course was satisfactory and the diabetes was brought under control. The patient was discharged 2 weeks after the second operation with minimal drainage from both ears.



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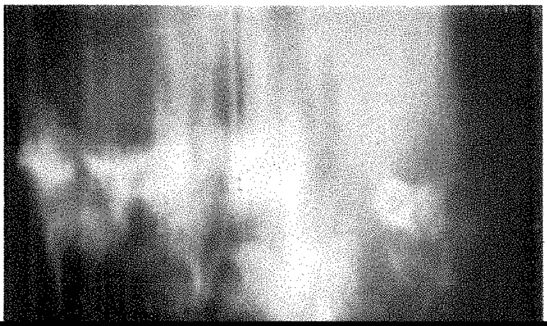

decade of life or older. All patients had a long-standing history of diabetes mellitus and most of them had complications such as septicemia, cardiopathy, nephropathy, etc. The clinical findings and symptoms were related to the extent of involvement. The symptoms consisted of pain, especially on manipulation of the external ear, purulent drainage, and swelling of the auricle and external auditory canal. Cases with an-

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examination allows an accurate evaluation of the extent and progression of the process.

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VOL. 112, NO. 2

RADIOISOTOPIC EVALUATION OF SALIVARY GLAND DYSFUNCTION IN SJÖGREN'S SYNDROME*

By DONATO ALARCÓN-SEGOVIA, M.D., M.S., YOLANDA GONZÁLEZ-JIMÉNEZ,
LUIS RENÉ GARZA, M.D., *and* JORGE MAISTERRENA, M.D.
MEXICO CITY, MEXICO

SALIVARY glands, like the thyroid gland, can selectively concentrate elements of Periodic Group VII and secrete them in saliva.[4,6] Scintillation scanning of salivary glands with technetium 99m (Tc^{99m}) pertechnetate has been developed utilizing this capability.[2,9,10,12–15]

Of the 3 components of Sjögren's syndrome (rheumatoid arthritis or other connective tissue disease, keratoconjunctivitis sicca and xerostomia), salivary gland dysfunction has been the most difficult to evaluate, particularly when early or mild. The radioisotopic studies presented herein were done to gain knowledge on the natural history and course of salivary gland dysfunction in Sjögren's syndrome. From them we have also developed a simple test for the diagnosis of subclinical xerostomia.

MATERIAL AND METHOD

Thirty-seven studies were done in 28 patients with Sjögren's syndrome in various stages of disease and treatment. Criterion for such diagnosis was the unmistakable presence of at least 2 of the 3 elements of the syndrome. Twenty had definite or classical rheumatoid arthritis, 2 had a clinical picture of rheumatoid arthritis but also had demonstrable LE cells, 1 had typical systemic lupus erythematosus and 1 gave the history of an episode of arthritis 2 years previously but had had no rheumatic symptoms since, despite detectable synovitis in both her knees. All 4 patients with xerostomia and keratoconjunctivitis sicca but without clinical connective tissue disorder had positive rheumatoid factor. One of them had active chronic hepatitis.

Twenty-one patients had noticed xerostomia and 2 more had acute salivary gland enlargement at the time of the study. There was or had been salivary gland enlargement in 4 of the 21 patients with xerostomia. Two other patients began noticing mouth dryness 45 and 60 days after the study, respectively.

All but 4 of the 28 patients had keratoconjunctivitis sicca as shown by slit lamp studies and Schirmer's tests.[5] All 4 had both a connective tissue disorder and salivary gland involvement.

Ten studies were done in as many healthy volunteers and served as controls.

Scintigrams were begun 30 minutes after the intravenous injection of 1.5 millicuries of technetium 99m eluted as sodium pertechnetate from a Mo^{99m}-Tc^{99m} generator* by flushing with sterile, pyrogen free isotonic saline. A Pho/Dot linear scanner† with a 3 by 2 inch crystal and a 61 hole collimator was used to scan the head and neck from the level of the eyes to below the thyroid.

Paper and photographic registries were done simultaneously. Anterior scans were done placing the collimator as close to the face as the nose permitted and leaving it at the same distance throughout the scanning. Following completion of the scanning, counts were made over each of the parotid glands, the roof of the mouth and the thyroid gland at the point of maximum uptake in each of these places as shown by the paper registry. The patient's head was then turned to the left and the collimator was placed 1 cm. over the cheek so as to allow it to run freely. This semilateral scan was

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† Nuclear Chicago, Des Plaines, Ill.

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From the Immunology Laboratory and Rheumatology Service and the Department of Nuclear Medicine, Instituto Nacional de la Nutrición, Mexico City, Mexico.

TABLE I

RADIOACTIVE COUNTS PER MINUTE OVER PAROTID GLANDS AND THYROID GLAND AND RATIO OF THYROID/PAROTID UPTAKE IN 28 SJÖGREN'S SYNDROME PATIENTS AND 3 SAMPLE CONTROLS

Subject	Date	Radioactive counts over			Ratio		
		Parotid		Thyroid	Thyroid/		
		Right	Left		Right Parotid	Left Parotid	Mouth*
Sjögren's Syndrome							
R.M.S.	2–10–70	16466	8735	94037	5.7	10.7	7.9
	2–25–70	11238	6435	34857	3.1	5.4	4.1
	4–6–70	14827	10911	58005	3.9	5.0	3.7
	6–20–70	11081	11671	73109	6.5	6.2	1.9
J.R.M.	2–18–70	7615	7828	18415	2.4	2.3	1.7
	3–30–70	13330	13276	12694	0.9	0.9	2.8
	5–8–70	8157	5655	10139	1.2	1.7	1.4
	7–13–70	11791	12691	28125	2.4	2.2	—
A.A.†	2–21–70	3050	3010	5001	1.6	1.6	1.1
	5–18–70	392	628	5900	15.0	9.3	0.9
I.W.R.	3–4–70	5127	3877	24359	4.7	6.2	5.9
	5–25–70	2680	2045	14908	5.5	7.2	2.0
N.A.V.	2–23–70	3762	3033	16395	4.3	5.4	—
	6–3–70	8479	5655	37031	4.3	6.5	8.6
R.B.G.	2–20–70	15559	15379	40858	2.6	2.6	2.9
R.H.S.†	2–23–70	2447	3926	7467	3.0	1.9	2.0
E.G.R.	3–3–70	9861	7648	16327	1.6	2.1	1.6
E.A.B.	3–4–70	5306	4852	48080	9.0	9.8	3.3
C.M.M.	3–6–70	6252	6048	18408	2.9	3.0	—
J.H.C.	3–9–70	5208	7344	29149	5.5	3.9	5.9
M.T.B.	3–16–70	4654	3900	38581	8.2	9.8	9.4
M.T.C.	3–16–70	3883	6210	29488	7.5	4.7	3.1
E.V.R.	3–16–70	8904	5735	22185	2.4	3.8	3.1
A.M.P.	3–17–70	4937	5112	105853	21.4	20.7	20.5
C.O.G.	4–2–70	9162	13559	21702	2.3	1.6	—
T.T.	4–7–70	6578	15922	17756	2.6	1.1	1.6
M.Z.F.	4–20–70	5756	12021	17394	3.0	1.4	1.1
M.E.P.	4–22–70	6001	6153	51384	8.5	8.3	8.4
N.H.M.†	5–25–70	5984	4872	5090	0.8	1.1	1.2
R.A.D.	6–5–70	2732	2565	19973	7.3	7.7	5.5
R.R.M.	6–6–70	3374	2778	53094	15.7	19.1	12.4
O.A.S.†	6–8–70	4928	3453	2975	0.6	0.8	0.7
A.E.P.	6–27–70	4954	5284	43037	8.6	8.1	4.1
V.M.M.	7–11–70	5788	3802	41665	7.1	10.9	3.1
H.B.T.	7–13–70	2820	2888	5707	2.0	1.9	1.6
A.C.	7–14–70	8069	7626	24645	3.0	3.2	5.1
R.R.E.	7–31–70	6927	8025	23667	3.4	2.9	0.8
Controls							
A.M.C.		8643	8915	14614	1.6	1.6	1.2
L.R.G.		15740	15141	29473	1.8	1.9	2.0
J.L.G.		7328	7243	17883	2.4	2.4	1.0

* Lateral scan.

† Hypothyroidism and/or Hashimoto's thyroiditis.

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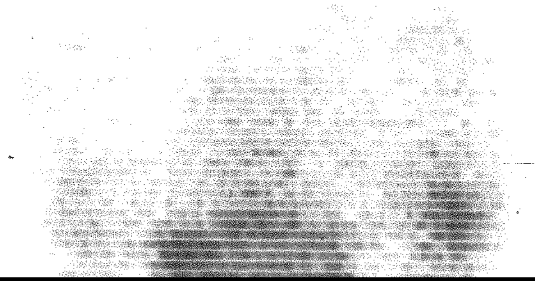

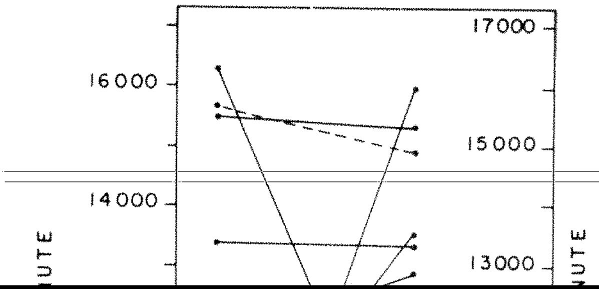

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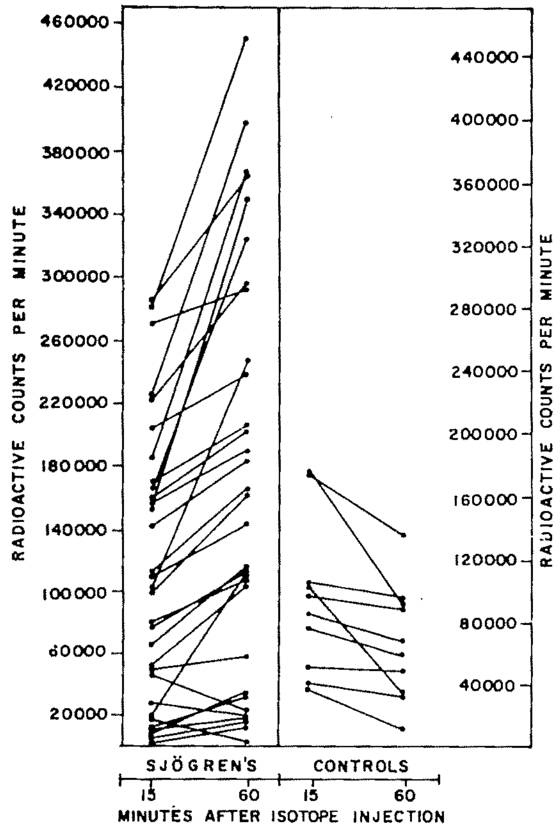

Fig. 6. Radioactive counts in saliva at 15 and 60 minutes after injection of Tc^{99m} pertechnetate in Sjögren's syndrome (31 studies) and normal controls (10 studies).

the changing pattern of salivary gland dysfunction in Sjögren's syndrome, as shown by repeated studies, brings new insight into its natural history and the feasibility of its being reversed either spontaneously or under treatment. These studies also allow recognition of salivary gland involvement prior to development of clinical xerostomia and may therefore afford detection of previously unrecognized cases of Sjögren's syndrome and, thereby, its better understanding.

The single measurement that allowed most definite differentiation between Sjögren's syndrome patients and normal controls is the rise in concentration of Tc^{99m} pertechnetate from the 15 to the 60 minute saliva samples. The 3 patients who had no such rise in their saliva had extreme and long-standing xerostomia and probably represent end stages of salivary gland damage involving the ductular apparatus. Concentration of Tc^{99m} pertechnetate in saliva at 15 and 60 minutes is a simple, rapid and inexpensive method for diagnosis and assessment of salivary gland dysfunction associated with Sjögren's syndrome.

SUMMARY

The capability of the salivary glands to selectively concentrate elements of Periodic Group VII and secrete them in saliva was utilized to assess salivary gland function in 28 patients with Sjögren's syndrome by means of radioisotopic studies following the intravenous administration of Tc^{99m} pertechnetate. The findings were compared with those obtained in 10 normal controls.

Scintiscans and direct counts over parotid glands showed lower uptake in patients than in controls. The counts were similar over both parotid glands in all controls, while there was significant difference of uptake between the two glands in many patients. Ratios of thyroid/parotid uptake were thus higher in most patients than in controls.

Concentration of the isotope in saliva diminished from the 15 to the 60 minute postinjection samples in all normal controls, while it increased in all but 3 of the patients. All three had extreme xerostomia and low salivary gland uptake.

Repeat studies in 5 patients revealed

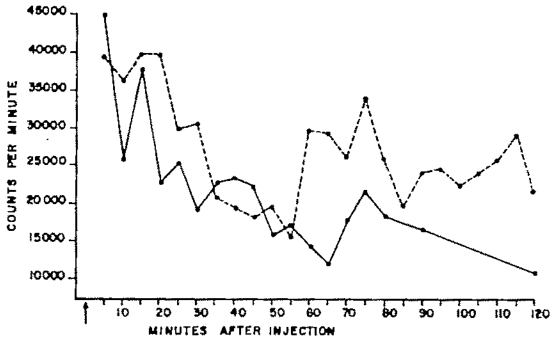

Fig. 7. Radioactive counts in the saliva of 2 normal controls following the intravenous administration of 1.0 millicurie of Tc^{99m} pertechnetate.

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ELEVATED SALIVARY IODINE AND SALIVARY GLAND ENLARGEMENT DUE TO IODINATED CONTRAST MEDIA[*]

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SALIVARY gland swelling following the intravenous administration of iodinated contrast media is a rare phenomenon, only 8 cases having been previously reported.[1,6,7,12] There have been additional isolated instances of sialadenopathy occurring after administration of bronchographic[10,18] and cholecystographic agents.[8]

We herein report a patient who developed submandibular salivary gland enlargement after each of 3 intravenous urographic examinations spaced over a 20 month period.

REPORT OF A CASE

A 57 year old male with bilateral polycystic kidney disease and advanced renal failure was recently started on a chronic hemodialysis program at the University Hospital of San Diego County. During the preceding 20 months he underwent intravenous urography on 3 occasions, October 1968, October 1969, and June 1970. He was azotemic at the time of all 3 examinations, the blood urea nitrogen values being 52, 94, and 124 mg. per cent, respectively. The contrast media used were: 1968—90 ml. of sodium and methylglucamine diatrizoate as renografin 60 per cent (approximately 26 gm. of organically bound iodine); 1969—90 ml. of sodium diatrizoate as hypaque 50 per cent (approximately 27 gm. of bound iodine); 1970—300 ml. of sodium diatrizoate as 25 per cent hypaque (approximately 45 gm. of bound iodine). No unusual reactions were experienced immediately following each of these studies, but on the third day postinjection each time, he developed bilateral, symmetric swelling of the submandibular salivary glands. The enlargement peaked at the fourth or fifth day, and gradually disappeared by the end of 1 week. In each instance there was only minimal nuchal discomfort and tenderness, with slight dysphagia and mild localized postprandial ache in the submandibular salivary glands. He had no skin rash, cough, coryza, excess lacrimation, nausea, or vomiting. Peripheral blood was not studied for the presence of eosinophilia. He has no other history of drug sensitivity, hay fever, or contact dermatitis. He has used topical iodine-containing solutions and has never demonstrated sensitivity to them.

On the third day following his most recent intravenous urography (June 1970), at a time when he manifested obvious submandibular salivary gland enlargement, samples of mixed saliva and heparinized blood were obtained for total iodine and inorganic iodide determinations. Total iodine was determined by chloric acid digestion of the sample to convert all iodine present to iodate, followed by reaction with iodide and titration with thiosulfate using starch as the indicator. For the inorganic iodide determination, the samples were first deproteinized with trichloroacetic acid. Addition of sodium nitrite converted any iodide present to iodine (I_2). Iodine was extracted into carbon tetrachloride and the carbon tetrachloride was titrated with thiosulfate. The results of these determinations are shown in Table 1. The plasma level of iodine corresponds to a plasma diatrizoate (hypaque) concentration of 186 mg. per cent.

DISCUSSION

Of the 8 previously described patients who developed salivary gland enlargement after urography, 3 were presumed to have normal renal function.[7,12] Of these, sodium diatrizoate was used in 2 patients,

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[†] James Picker Foundation Scholar in Radiological Research.

Table I

IODINE CONTENT OF PLASMA AND SALIVA 3 DAYS AFTER INJECTION OF 300 ML. OF 25 PER CENT SODIUM DIATRIZOATE

	Total Iodine (mg. per cent)	Iodine as Iodide (mg. per cent)
Plasma	112	0
Saliva	11	4

and sodium and methylglucamine diatrizoate in the third. Two patients cited by Ansell[1] had parotid gland swelling after infusion urographies but other details are not known. Harris *et al.*[6] recently reported 3 patients who developed submandibular salivary gland swelling after high dose urography, but data on renal function were not given.

Since salivary gland swelling is an infrequent but recognized manifestation of iodism or idiosyncrasy to inorganic iodides,[3] it is logical to ask whether the sialadenopathy due to iodine-containing contrast agents is mediated by free iodide. There is, to date, no direct evidence that the iodine in contrast media, which *in vitro* is tightly bound to the organic ring, will be split off from the molecule *in vivo*. However, accurate direct measurements of very small amounts of inorganic nonradioactive iodide in the plasma are difficult, if not impossible, to obtain.

Human salivary glands, like the thyroid gland, are known to concentrate iodide up to 100 times the plasma inorganic iodide level.[5] To date, however, no organic iodine has been reported in salivary excretions, even when organically bound iodinated compounds are circulating in the blood and excreted in the urine.[4,9]

The surprising finding of large amounts of total iodine and free iodide in the saliva of this patient leads to interesting speculation about the salivary gland handling of contrast media. Three days after the infusion of 300 ml. of 25 per cent hypaque, this patient with severe renal failure had a plasma total iodine concentration of 112 mg./100 ml., corresponding to a plasma diatrizoate concentration of 186 mg./100 ml. Simultaneously, the saliva contained 7 mg./100 ml. of organically bound iodine. The latter most likely represented intact diatrizoate, although this would have to be proved. (An attempt was made to document the presence of diatrizoate in the saliva by U.V. spectrophotometric analysis using the known absorption of diatrizoate at 238 μ,[11] but the patient's saliva contained interfering substances in this range of the ultraviolet spectrum.)

The chemical determinations of inorganic iodide in the saliva revealed the markedly elevated concentration of 4 mg./100 ml. If one assumes a maximum concentrating ability for the salivary glands of 100 times the Plasma Inorganic Iodide level (PII),[5] the PII, by extrapolation, would be elevated to at least 40 μg./100 ml., a concentration nevertheless beneath the detectability potential of nonradioactive assays. Although uremic patients on normal diets are known to have an elevated PII, the mean values determined by radioactive assay are only in the range of 1–2 μg./100 ml.[2] Even though there was a presumptive elevation of PII and a known elevation of salivary inorganic iodide in our patient at a time when he manifested signs of iodism, one cannot exclude the possibility that the intact contrast molecule itself caused the salivary gland enlargement.

In the presence of renal insufficiency, nonrenal routes of contrast media excretion become quantitatively significant. Opacification of the gallbladder and intestines is frequently observed after intravenous urography in such patients. It appears now, that at least one other extrarenal route of excretion exists; *i.e.*, via the salivary glands. We are currently measuring plasma and salivary iodine and iodide concentrations in a series of patients receiving intravenous contrast media to further study some of these questions.

SUMMARY

After each of 3 intravenous urographic examinations a patient with advanced renal

failure developed salivary gland enlargement.

Total iodine and free iodide concentrations were measured in mixed saliva and in plasma collected while he had sialadenopathy.

These measurements revealed high concentrations of both organically bound iodine and free iodide in the saliva at a time when the plasma diatrizoate level was high.

The findings suggest secretion of contrast media by the salivary glands, concomitant with the secretion of free iodine split off of the organic contrast molecule.

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THE RADIOLOGY OF THE HUGHES-STOVIN SYNDROME*

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IN 1959, Hughes and Stovin[3] described a syndrome of venous thromboses occurring in the legs, vena cava or right heart

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areas of underperfusion peripherally in both lungs. An inferior vena cavagram was normal. A selective right pulmonary angiogram (Fig. 2)

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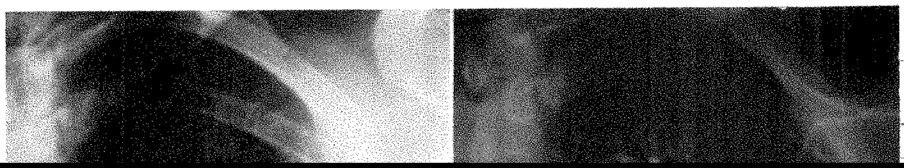

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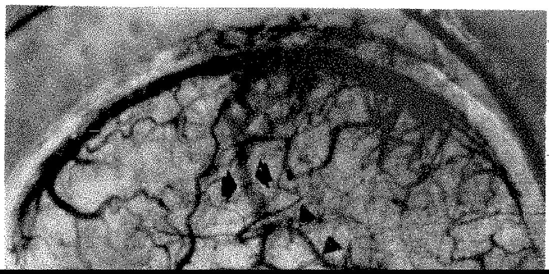

The etiology of the syndrome remains obscure. Infective emboli,[6] collagen disease,[4] congenital anomalies[3] and polyarteritis nodosa[4] have all been implicated. Hughes and Stovin[3] considered that degenerative changes in the bronchial arteries in their cases led to inadequate nutrition of the pulmonary arteries with resultant aneu-

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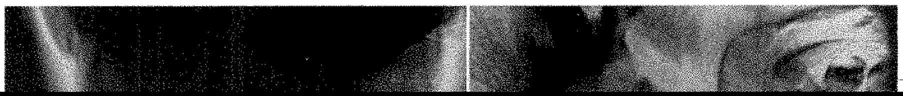

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BILATERAL NEUROFIBROMA OF THE INTRATHORACIC VAGUS ASSOCIATED WITH VON RECKLINGHAUSEN'S DISEASE*

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NEUROGENIC mediastinal tumors are not uncommon and comprise the majority of neoplasms of the posterior mediastinum. Those arising from the vagus nerve, however, are rare and may present as a mass elsewhere in the mediastinum. In a review of the literature Gayola *et al.*[4] found 19 cases of intrathoracic vagus sheath tumors previously reported, and the authors added an additional case of their own. Of these 20 cases, 12 were classified as neurofibromas,[1–6,8–10,13] and, of these, 6 were definitely associated with von Recklinghausen's disease.[1,3,5,9,10] In all but one reported case,[6] involvement of the vagus nerve was unilateral.

This report adds another case and represents the first example of bilateral neurofibroma of the intrathoracic vagus nerve in a patient with generalized neurofibromatosis or von Recklinghausen's disease.

REPORT OF A CASE

K.W., an 18 year old soldier, was seen on sick call in Fort Riley, Kansas, in November 1969 for low back pain. At this time subcutaneous nodules were noted in the upper and lower extremities and in the back and on the forehead, with pigmented lesions characteristic of café-au-lait spots on the trunk. In July 1970 the patient was next seen at U.S. Army Hospital, Fort Ord, where the above described subcutaneous nodules and pigmented spots were again noted. Physical examination of the patient was otherwise negative. Laboratory studies were normal as were skeletal and skull roentgenograms.

An excisional biopsy of a subcutaneous nodule on the right thigh was performed on July 29, 1970; the pathologic report was plexiform neurofibroma.

On the routine preoperative chest roentgenogram, a bilateral superior mediastinal mass was noted, most marked on the left side, adjacent to and confluent with the aortic arch (Fig. 1, *A* and *B*); tomography delineated the mass in greater detail (Fig. 1*C*). On August 24, 1970 a left thoracotomy was performed, and the mass at the aortic arch was found to be part of the vagus nerve which was beaded and nodular in appearance. The left recurrent laryngeal nerve was also involved. The mass as well as the left recurrent laryngeal nerve was resected. The surgical specimen was soft and nodular and measured 11 cm. in length varying from 0.4 to 2.2 cm. in diameter (Fig. 2). The histologic report of the surgical specimen was neurofibroma of the vagus nerve (Fig. 3). The right thorax was not explored, and, except for a mild degree of hoarseness, the postoperative course was uneventful (Fig. 1*D*).

DISCUSSION

Neurofibromatosis (von Recklinghausen's disease) is a congenital and hereditary disease and clinically exhibits multiple cutaneous nodules, patches of cutaneous pigmentation (café-au-lait spots), and neurofibromas of peripheral nerves. About 51 per cent of affected patients exhibit skeletal lesions or deformities.[7]

In the chest, neurofibromatosis may be manifested as a thoracic mass. The neurofibroma usually arises from the sheath of the root of a spinal nerve or from the sympathetic chain; it may be confined to the spinal cord, or it may extend into the chest

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* From the Radiology Service, US Army Medical Department Activity, Fort Ord, California.

This material has been reviewed by the office of the Surgeon General, Department of the Army, and there is no objection to its presentation and/or publication. This review does not imply any indorsement of the opinions advanced or any recommendation of such products as may be named.

The views of the authors do not purport to reflect the position of the Department of the Army or the Department of Defense.

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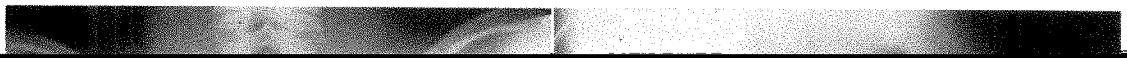

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racic mass and is indistinguishable from an intrathoracic neurofibroma on the plain roentgenogram.[11]

Of the reported cases of neurofibromas of the vagus nerve, only one has been bilateral,[6] but this was not associated with generalized neurofibromatosis or von Recklinghausen's disease. The present case represents the first case of bilateral neurofibroma of the vagus nerve associated with von Recklinghausen's disease.

Neoplasms of the intrathoracic vagus nerve, like many others in the mediastinum, usually are asymptomatic and are discovered only on routine chest roentgenograms. Dysfunction attributable to involvement of vagal fibers to the heart, bronchial musculature, or gastrointestinal tract has not been noted preoperatively in either our case or those in the literature. Also, like many other mediastinal neoplasms, neurofibrom~~a of the intrathoracic~~

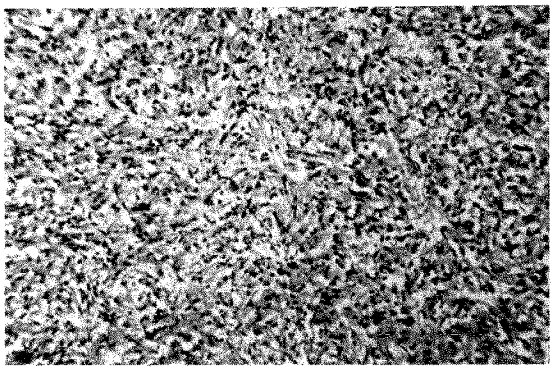

Fig. 3. Photomicrograph of a section of the surgical specimen showing characteristic appearance of a neurofibroma. The tumor is composed of wavy fibrils of young collagen. Embedded among the wavy fibers are large numbers of nuclei that are oval to spindle shaped and uniform in size; only occasionally do they lie in parallel rows.

Th~~is appears to be the first reported case~~ ~~associated with generalized neurofibroma~~tosis or von Recklinghausen's disease.

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NEUROGENIC PULMONARY EDEMA*

OBSERVATIONS IN 6 PATIENTS

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PULMONARY edema occurs clinically in association with a variety of central nervous system abnormalities including convulsions, trauma, hemorrhage, and neoplasm. Artificial elevation of intracranial pressure as well as head trauma, and other central nervous system insults have been used experimentally to induce similar lung changes.[2,5,6,8–10,13]

The roentgenograms of 6 patients, 4 children and 2 adults, who developed pulmonary edema as a result of intracranial abnormalities, have recently been studied at the University of Florida Hospital. While this association has been reported in the medical literature,[1,2,4–10,12,13] scant attention has been paid to it in the radiologic literature.[3,12] Since pulmonary edema may first manifest itself roentgenographically, awareness of this complication in patients with central nervous system disease is necessary to ensure proper treatment.

CLINICAL AND ROENTGENOGRAPHIC FINDINGS

Table I summarizes the clinical and roentgenographic features in these 6 cases.

In Cases I and II (Fig. 1, *A* and *B*) pulmonary edema developed in association with severe, fatal head trauma. Case III (Fig. 1*C*) sustained milder head trauma followed by complete recovery.

Cases IV, V, and VI developed pulmonary edema in association with increased intracranial pressure. Involvement was predominantly on the right side in 2 (Fig. 2*A*; and 3*A*) and exclusively right-sided in the third (Fig. 4*A*). The cause for this peculiar right-sided distribution is unclear at present. In Cases IV and V the roentgenographic

TABLE I

SUMMARY OF CASES

Case	Age Sex	Central Nervous System Abnormality	Course of Illness	Roentgenographic Findings	Outcome
I. D.T.	18 yr. F	Temporoparietal skull fracture	Admitted in coma and decerebrate rigidity. Blood pressure 125/80, Pulse 88. 100 cc. epidural hematoma evacuated	Normal chest roentgenogram on admission. Diffuse pulmonary edema developed within 6 hours	Expired after 24 hours. No autopsy
II. T.H.	10 yr. M	Gunshot wound of right temporoparietal area	Comatose and areflexic on admission. Blood pressure 120/70, Pulse 120	Diffuse pulmonary edema—predominantly of the upper lobes	Expired after 10 hours. No autopsy
III. G.J.	7 yr. M	Head trauma	Stuporous but not unconscious. Left and right-sided seizure followed by uneventful recovery. Blood pressure 110/46, Pulse 100	Upper lobe pulmonary edema—predominantly left-sided. Cleared in 24 hours	Uneventful recovery
IV. D.G.	7 yr. F	Cerebellar astrocytoma with secondary hydrocephalus	Headaches for 6 months. Two seizures prior to admission. Blood pressure 90/70, Pulse 140. Cerebrospinal fluid pressure 300 mm. H_2O	Cranial sutures widened, ventricles dilated. Pulmonary edema present on admission, predominantly right-sided. Chest clear 2 days following craniotomy	Tumor resected. Alive 8 months
V. C.B.	22 mo. M	Post meningitic hydrocephalus. Cardiac end of V-A shunt placed in right ventricle inadvertently	Admitted for increasing lethargy and ataxia. Pulse 120. Cerebrospinal fluid pressure over 300 mm. H_2O	Cranial sutures widened. Right-sided pulmonary edema cleared following shunt revision	Alive and well 4 months
VI. P.S.	9 mo. F	Acute myelomonocytic leukemia with infiltration of the meninges	Admitted for treatment of leukemia. Following initial steroids, patient received allonrinol, thioquanine, and cytosine arabnoside	Cranial sutures markedly widened. Right-sided pulmonary edema developed after 3 days and resolved 24 hours later	Alive 3 months

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of the vagus nerve in neurogenic pulmonary edema, both clinically as well as in laboratory animals.[2,5,6,9] In some animal experiments, neurogenic pulmonary edema could be prevented by vagotomy prior to experimental trauma.[9] Campbell *et al.*[2] considered atropine helpful in reversing neurogenic pulmonary edema in experimental animals. Harrison and Liebow[6] suggest that atropine may provide a period of grace during which operative intervention may relieve the rising intracranial pressure. However, Ducker and Simmons[5] were not able to prevent neurogenic pulmonary edema by previous cervical vagotomy in animals subjected to increased intracranial pressure.

The parenchymal distribution of neurogenic pulmonary edema is unpredictable. Richard[11] found unilateral and bilateral upper lobe edema common among his patients. Rigler and Surprenant[12] illustrate a patient with isolated left lower lobe edema. The 11 cases reported by Ducker[4] had generalized distribution.

The upper lobe distribution in our 3 patients with trauma and the isolated right-sided localization in those with chronic increased pressure are noteworthy, but no definite conclusion is apparent. Positional factors may be important.

The treatment of patients with neurogenic pulmonary edema should be directed toward prevention and control of seizures and relief of increased intracranial pressure when present. Atropine may be of help in some patients and oxygen and other supportive measures should be employed.[6] Digitalis, however, may not help and morphine derivatives are contraindicated since they are known to increase intracranial pressure.[7] Intravenous fluid should be administered with care so as not to increase the load on an already elevated pulmonary venous system.

SUMMARY

Patients who sustain head trauma, with or without seizures, or who develop increased pressure for other reasons should be suspect for the development of pulmonary edema. Early recognition may avoid confusion with congestive heart failure. This distinction is of importance since the usual drugs used in the treatment of congestive heart failure are likely to increase the severity of neurogenic pulmonary edema.

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VERTEBRAL SCALLOPING IN ACROMEGALY*

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THE roentgenographic spinal changes in acromegaly are well established.[3,7,11] However, "scalloping," that is, exaggeration of the normal slight concavity of the posterior surface of the vertebral bodies, has not been emphasized. The observation of this finding in a number of acromegalic patients stimulated a complete review of our material for the purpose of documenting the occurrence of scalloping in acromegaly and defining its incidence.

MATERIAL AND METHOD

Roentgenographic material from 38 well-documented acromegalic patients admitted to the University of Kansas Medical Center between 1955 and 1969 was reviewed. Lateral lumbar spine roentgenograms were evaluated and the degree of scalloping graded as minimal, moderate and marked. Lateral chest roentgenograms or lateral thoracic spine studies were also graded, since in some of the patients these were the only available films. The interpediculate distance of the third lumbar vertebral body was measured in an attempt to correlate the degree of scalloping with enlargement of the spinal canal.

Slight physiologic scalloping is frequently present in the population at large; therefore, 50 random lumbar spines were studied as a control group. The presence or absence of scalloping was recorded, and the interpediculate distances were measured.

Table I

OCCURRENCE OF THORACIC AND LUMBAR VERTEBRAL BODY SCALLOPING IN 38 ACROMEGALIC PATIENTS

1. Moderate or marked scalloping	15/38 or 39 per cent
2. Minimal scalloping	12/38 or 32 per cent
3. No scalloping	11/38 or 29 per cent

Table II

INCIDENCE OF SCALLOPING IN THE 17 ACROMEGALIC PATIENTS WITH LATERAL ROENTGENOGRAMS OF THE LUMBAR SPINE

1. Moderate or marked scalloping	11/17 or 65 per cent
2. Minimal scalloping	6/17 or 35 per cent
3. No scalloping	0 per cent
4. Total scalloping	100 per cent

RESULTS

Of the 38 acromegalic patients, 15, or 39 per cent, had moderate or marked scalloping (Fig. 1, *C* and *D*; Table I). If the 17 patients with roentgenograms of the lumbar spine are considered separately, moderate or marked scalloping was demonstrated in 65 per cent and minimal scalloping in 35 per cent; that is, some degree of scalloping existed in all the patients examined (Fig. 1; Table II). Of the 21 patients who had only roentgenograms of the thoracic spine, 14 per cent showed moderate scalloping, and a total of 48 per cent demonstrated some degree of scalloping (Fig. 2; Table III).

Forty-four per cent of the control group showed some degree of scalloping (Fig. 1*A*; Table IV). In 3 patients the degree was considered to be moderate. None of the 50 control patients showed a marked degree of scalloping.

No statistically significant correlation between scalloping and widening of the interpediculate distance was found.

DISCUSSION

Scalloping or erosion of the posterior vertebral bodies has been described in a wide variety of pathologic entities, including congenital enlargement of the spinal

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tients. However, scalloping of the vertebrae as a common acromegalic feature has not been emphasized.[1,2,6,12]

The incidence of scalloping of vertebral bodies in acromegalic patients appears to be much higher than the previous literature would indicate. This finding may have been overlooked in the past because of the emphasis on other spinal changes or because lumbar spine roentgenograms were not available. In general, the scalloping was more severe in those patients with long-standing vertebral disease. A few patients demonstrated scalloping who had had a relatively short clinical course. The scalloping did not decrease following arrest of the disease.

The pathogenesis of these spinal changes is difficult to ascertain. There is a paucity of pathologic material. An attempt was made to evaluate our autopsy material, but unfortunately the spine was usually not thoroughly examined. Also, the dura and connective tissues in the spinal canal were not specifically examined.

Steinbach and associates[11] theorized that scalloping could be secondary to pressure erosion from enlarged soft tissues, or could be due to modeling and resorption as a direct result of elevated growth hormone levels. Any hypothesis must take into account the permanence of the bony lesions, even after the arrest of the disease, and the preponderant involvement of the lumbar spine. One of the striking findings in acromegaly is the exuberant hyperplasia of soft tissue structures.[8,12] Hyperplasia of the soft tissues of the spinal canal could cause scalloping but does not explain why this sign is uncommonly seen in the thoracic region. There is some regression of soft tissue hyperplasia following arrest of the disease, but the scalloping does not improve as one might expect. Connective tissue hyperplasia should cause generalized enlargement of the spinal canal, but the interpediculate distances were not significantly widened in this series. Selective modeling and resorptive changes are seen in the extremities, particularly in the metatarsals. It would not be unreasonable to assume that selective bony resorption causes the scalloping. Since the other bony changes in acromegaly are permanent, one would not expect the scalloping to regress with the return of growth hormone levels to normal.

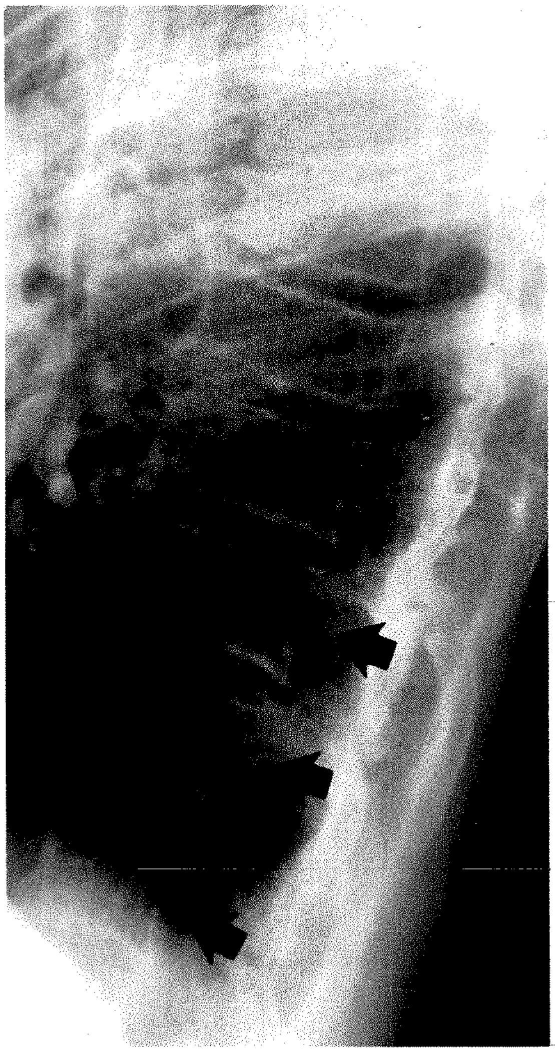

Fig. 2. Lateral view of the dorsal spine in an acromegalic patient revealing scalloping in the lower portion (arrows).

SUMMARY

Roentgenograms of the thoracic and lumbar spine of 38 acromegalic patients were evaluated to determine the frequency of posterior vertebral body scalloping.

Scalloping was found in a significant

number of patients, particularly in the lumbar region. The occurrence of this sign has not been adequately emphasized in the past.

Possible pathogenic mechanisms are briefly discussed.

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DEVELOPMENTAL STENOSIS OF A THORACIC VERTEBRA RESULTING IN NARROWING OF THE SPINAL CANAL

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A REVIEW of the medical literature on stenosis of the spinal canal discloses, to the best of my knowledge, that reports published up to the present have dealt only with lesions involving the cervical or the lumbar regions.

A case of spinal canal stenosis involving a thoracic vertebra came recently to our observation. It apparently is the first to be reported.

HISTORY

In 1953, Schlesinger and Taveras[13] pointed out that a narrow spinal canal in patients with herniated disks could produce typical clinical findings and symptoms suggesting multiple root and even cauda equina compression.

Pallis *et al.*[10] in 1954, in anatomic and roentgenologic studies of the cervical spine, found that a narrowing of the spinal canal could be produced, among other causes, by "osteophytes arising at the posterior borders of the vertebral bodies." Payne and Spillane[11] in a later study discovered that small diameters were found in spondylotic spines, and it was felt that "the development of myelopathy may also be related to the initial size of the canal."

In 1963, Epstein *et al.*[5] described a case of osteophytes in a cervical vertebra in which at surgery an unusual narrow spinal canal was found.

Verbiest[14,15] demonstrated that a minus variation in the diameter of the spinal canal in the lumbar segment could result in a compression of the roots of the cauda equina. This author regarded the stenosis of the spinal canal as a localized and limited developmental abnormality.

Gill and White[8] reported that in a few patients with lumbosacral transitional vertebrae there was a noticeable reduction in the anteroposterior diameter of the spinal canal, almost as small as the diameter of the normal sacral canal.

Friedman[7] found that in a few cases of narrow lumbar spinal canal, a marked thickening of the neural arches was present.

Epstein *et al.*[4] in a study of low back pain and radiculitis noticed that in a few individuals the symptomatology and the clinical findings did not always conform to the typical pattern related to the compression syndrome produced by osteophytes or herniated disks.[8] Preoperative myelography demonstrated single or multiple defects and at times a complete block.

REPORT OF A CASE

G.H. G705, female patient, 67 years of age. The present illness goes back to 3 months prior to admission, when the patient noticed that on walking she was dragging her left foot. This symptom did not persist. However, since that time she had to watch how and where her feet were moving. On admission she stated that she had a "numb, heavy feeling" from her middle thoracic spine down both legs.

The clinical examination showed normal fundi, and no abnormalities involving the cranial nerves. Neurologically, the upper extremities were normal. At the level of the upper quadrants of the abdomen both temperature and pain sensation showed a decrease in intensity. The upper abdominal reflexes were present, but absent in the lower abdomen. Both knee and ankle jerks were exaggerated bilaterally and there was spasticity of both lower legs, more on the right, with marked adductor spasm. Uncontrolled, spontaneous muscle contractions of the lower extremities were also present, together with loss of position sense.

Roentgen studies of the thoracic and lumbar

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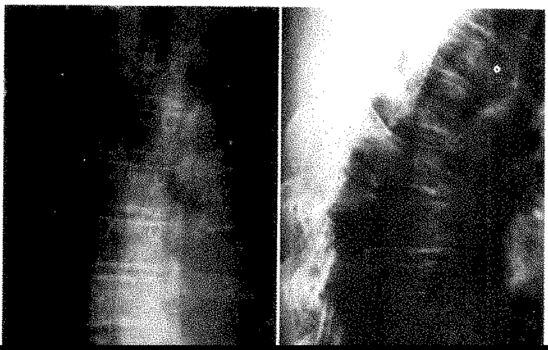

vertebrae showed diffuse spondylosis (Fig. 1, *A* and *B*), and diskogenic disease in the lower lumbar segment. A correct evaluation of the interpedicular distance, of the transverse and of the anteroposterior diameters of the thoracic vertebrae, in accordance with the method adopted by Jones and Thomson[9] was not possible because of a slight rotation of the thoracic vertebrae with minimal scoliosis. Similarly, we could not correctly evaluate the interpedicular diameter with the method proposed by Elsberg

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with the cases reported in the literature and which deal only with spinal canal stenosis in the cervical and lumbar vertebrae.

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HEREDITARY MULTIPLE EXOSTOSES*

REPORT OF A CASE WITH SPINAL CORD COMPRESSION

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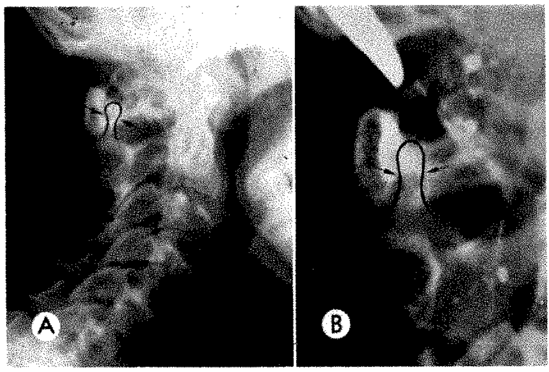

FIG. 2. (*A*) Lateral cervical spine roentgenogram. A tubular excrescence is faintly visualized arising from the arch of C-2 and extending superiorly anterior to the neural arch of C-1. There is a suspicion of widening of the distance between the lateral aspects of the neural arches of C-1 and C-2. (*B*) LogEtron of lateral cervical spine at myelography. The exostosis can be readily appreciated arising from the arch of C-2.

forming chondrodysplasia) was first described by Stanley in 1849.[8] This condition usually becomes manifest during childhood when bumps on the skeleton are discovered by the patient or a relative. The long bones are most frequently involved, although exostoses may form from any bone preformed in cartilage. Although Jaffe[8] states that the development of chondrosarcoma in multiple exostoses is common (up to 20 per cent), Lichtenstein[10] feels that it occurs in only 1 to 2 per cent of cases. A family history of this disorder can be elicited in 65 per cent of the cases.[8] In the remainder the condition arises spontaneously, as in the case reported here.

Involvement of the spine is frequent in patients with multiple exostoses (7 per cent in one series),[3] but associated spinal cord compression is rare. In a 1951 review, Slepian and Hamby[13] found only 9 cases in the literature and added one of their own. Since that time 8 additional cases have been recorded.

Most cases[1,2,4–7,9] have involved compression of the spinal cord in the thoracic or lumbar area, with only 4 tumors involving the cervical spine.[3,11–13] The age range of the patients previously reported is 13 to 33 years at the time of diagnosis. Symptoms are those of a slowly growing spinal cord tumor.[4]

Published roentgenograms of exostoses in the thoracic and lumbar spine show striking abnormalities. The exostoses are large and cauliflower-like, extending the length of two or more vertebral bodies. The large size of these tumors makes exact localization of their origin difficult. In 2 recent case reports[4,6] the lesions producing spinal cord compression arose at the costovertebral junction.

Of the 4 previously reported cases of cervical exostoses with spinal cord compression, 2 included roentgenographic findings. The exostoses in Slepian and Hamby's case[13] presented as a neck mass, and roentgenograms revealed a large knobby exostosis arising from C-5 or C-6. In our patient a thin tubular bony excrescence was seen arising from the neural arch of C-2 and extending cranially beneath the neural arch of the atlas. The visualized bony pedicle of the exostosis belied the actual size of the

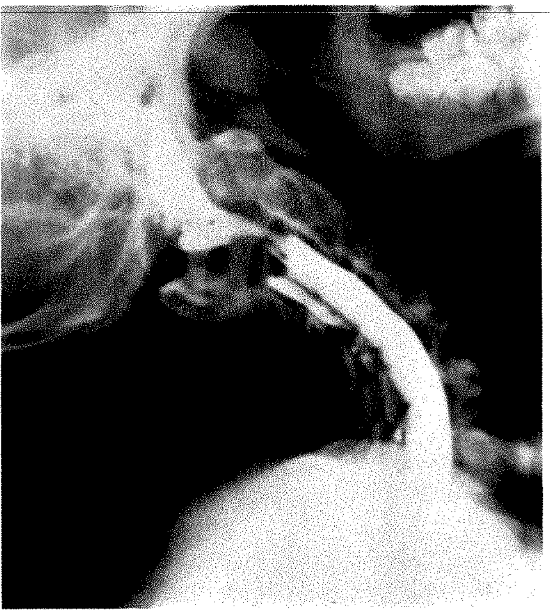

FIG. 3. Lateral cervical spine at myelography. A posterior extradural defect in the pantopaque column at the C-1 C-2 level is seen. The size of the defect reflects the presence of the cartilaginous cap of the exostosis.

tumor (as can be appreciated by the relatively large extradural defect on the myelogram) because the cartilaginous cap of the exostosis had insufficient calcium within it to be roentgenologically visible. In 1968, Carmel and Cramer[3] reported a case of an exostosis of C-2 in a 13 year old girl with roentgenographic findings similar to those in the case reported here.

SUMMARY

A case of hereditary multiple exostoses in which spinal cord compression by a neural arch exostosis occurred is reported in a 14 year old female.

An exostosis impinging on the spinal cord is an unusual complication of this disease, but the radiologist should be aware of its possibility. In the patient with spinal cord signs, the offending exostosis should be diagnosed with appropriate roentgenograms and myelographic investigation.

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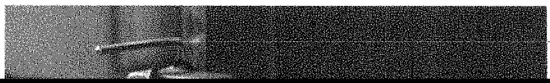
SKULL IMMOBILIZER AND POSITIONER*

By RUBEM POCHACZEVSKY, M.D., *and* SAMUEL LEVINE, R.T., L.X.T
BROOKLYN, NEW YORK

PROPER immobilization of the skull for cerebral arteriography is of the utmost importance since the slightest deviation from the proper position can be misleading and complicates interpretation of the arteriogram.[1] Malpositioning may result in the need for repeated injections with the attendant hazards of increased dose of radiopaque medium and prolongation of examination time. Skull immobilization, however, for prolonged periods of time, particularly in uncooperative or semicomatose patients is not an easy task.

Several immobilization techniques have been used including securing the head with adhesive tape and vacuum head clamps. Adhesive tape is clumsy to use, uncomfortable to the patient and inadequate. Vacuum head clamps require a large smooth surface for the mounting of the vacuum cups. Since they are very heavy, they tend to fall when released and are often unreliable. In addition, head clamps cannot

gles in order to bring the patient's orbitomeatal line perpendicular to the film or to any other desired angulation. This adjustment is made by simply moving a sustaining cylinder bar to different slots in the Immobilizer walls. The chin can be fixed by means of another movable cylinder bar running across the front of the Immobilizer (Fig. 2, *A–D*). Inflatable plastic bladders or sponges are used to prevent lateral motions or rotation of the head within the Immobilizer. We prefer to use inflatable bladder cushions, since they are disposable and can easily be inflated with air. They have built-in valves which make the use of a clamp unnecessary. They can also be easily inflated by means of a syringe with an adapter or by means of a CO_2 tank. The Immobilizer has a flat back which allows it to rest firmly on



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the serial changer or radiographic table. For the usual cerebral arteriogram, the head is positioned with the orbitomeatal line perpendicular to the film and the tube is angled 15° toward the feet with the central ray entering 2 inches above the glabella and exiting at the occipital protuberance.

ADVANTAGES

The following are the advantages of the Skull Immobilizer and Positioner:

It is more comfortable for the patient since his head rests, fully supported, on a flat inclined shelf.

Immobilization is accomplished quickly and the head's position can be easily readjusted at any time during the procedure.

Effective immobilization of the patient's head facilitates puncture of the carotid artery without recourse to additional personnel to position uncooperative patients.

Avoids crude methods of immobilization such as strapping or the need to use cumbersome vacuum clamps.

The unit is completely transparent and allows unobstructed observation of the patient's eyes, lips and face.

It is completely radiolucent.

It allows the use of inflatable bladder cushions which are more comfortable and effective.

Since the angulation of the head is easily accomplished with this device, it is possible to decrease or eliminate the angulation of the x-ray tube if desired. This is important when there is limitation of space or when special grids are used, precluding angulation of the tube.

SUMMARY

A new, easy to use, transparent, radiolucent Skull Immobilizer and Positioner, with built-in angulator, is described.

Its use is advocated for cerebral angiography.

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REFERENCE

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Fifty-fourth Annual Meeting: Boca Raton Hotel and Club, Boca Raton, Florida, May 14–19, 1972.

* Deceased.

EDITORIALS

FIFTY-THIRD ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE Fifty-third Annual Meeting of the American Radium Society was held in Mexico City at the María Isabel Sheraton Hotel, March 15 to March 18, 1971. A total registration of 666 was recorded, representing 219 members and the remainder included guests, ladies and residents in training. This attendance was one of the largest in the history of the Society, attesting to the quality of the Scientific Subjects and the attraction of Mexico.

Dr. James F. Nolan officially called the Meeting to order on Monday morning, March 15 at 8:30 A.M. He introduced Dr. Angel Matute, President of the Academia Mexicana de Cirugía, who gave the welcoming address in Spanish. Dr. José Noriega was gracious enough to translate the talk into English. He outlined the ancient and colonial history of Mexico and stressed the importance of communicating knowledge rapidly in modern times.

Dr. Nolan continued the meeting with his Presidential Address with the theme of the "Risks and Hazards in the Delivery of the Product." He related his own personal history in the practice of oncology since he attended his first national medical meeting in which he participated. This meeting was that of the American Radium Society at Atlantic City in June, 1942. He has been a provider of medical care with exposure to ionizing radiations for nearly 25 years. The total body radiation received in a 20 year period was approximately 50 rem without physiologic effects which could be attributed to radiation injury. He performed a total of 2,029 radium applications between the years of 1950 and 1970. Other types of risks and hazards attributable to practice, but not to radiation, are stress and strain to the coronary circulation and to the gastric mucosa. The hazard of the malpractice suit has only resulted in depositions and the threat of a court appearance. Improved techniques in radiation protection and radiation delivery, advances in surgical techniques, the availability of hormones and chemotherapeutic agents and the development of more efficient systems of delivery of quality health care will more than compensate for the risks and hazards.

The First Scientific Session, under the Chairmanship of Dr. John V. Blady, Philadelphia, Pennsylvania, began with a *Symposium on Early Carcinoma of the Breast.* The symposium was moderated by Dr. William E. Powers of St. Louis who introduced the various speakers on the panel. Dr. H. Stephen Gallagher showed serial whole organ sections to demonstrate multiplicity of invasive nodules along the path of a duct. He indicated that hyperplastic epithelium either remained as hyperplastic tissue, developed into carcinoma or became non-malignant lesion such as papilloma. Dr. John E. Martin of Houston pointed out that baseline studies of the breast with serial examinations over intervals of time from 6 months to 2 years have provided valuable early information on developing cancers within the breast. Dr. William Donegon from Columbia, Missouri favored elective biopsy to high risk population and showed statistically a high yield of subclinical cancer in the second breast when employed for patients with lobular cancer. Dr. Aaron G. Fingerhut emphasized the team effort at the new tumor clinic at the Los Angeles County-University of Southern California Tumor Center. He discussed the possibility of using excisional biopsy of suspected lesions or wedge resection of known probable lesions followed by radiotherapy of the

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has proven to be most helpful in initial staging of patients and subsequent management. Dr. Charles H. Taggart *et al.* from M. D. Anderson Hospital in Houston gave a description of interstitial and external radiotherapy techniques in a series of 37

After the intermission, Dr. Hans-Ludvig Kottmeier of the Radiumhemmet of Stockholm related his experience in diagnosis and treatment of carcinoma of the endometrium. He employs fractional curettage, biopsy, lymphography, hysteroscopy and hysterosalpingography in determining the details of precision diagnosis. The size and length of the uterine cavity, and especially the microscopic type of lesion, influence the treatment. In 1,300 cases treated from 1961 to 1965, the over-all survival rate was 59 per cent and the actual cure rate was 50 per cent.

Other papers presented in the latter part of the morning session included those of Dr. John M. Molendyk *et al.* on adjunctive radiation therapy for breast carcinoma, Dr. Norman Simon on a survey of afterloading radiotherapy systems with the use of cesium 137, and Dr. Bernard Roswit *et al.* on radiation injuries of the stomach, small bowel and colon.

The highlight of individual presentations of the meeting was at the close of the Tuesday session when the Janeway Lecture was delivered by Dr. Lauren V. Ackerman of Washington University School of Medicine of St. Louis. The title of the address was "The Pathology of Irradiation Effect in Normal and Neoplastic Tissue." Following a brief review of his early experiences as a pathologist, mingled with interesting anecdotes, he related the gradual evolution that took place in his own thinking processes in correlating the effects of irradiation in normal and neoplastic tissue. He emphasized that the pathologist must have certain critical information relating to details of radiotherapy technique in order to assess the status of tissues following treatment. He thought that the most important factor in radiation therapy was to preserve as good a blood supply to the irradiated tissue as possible, for if there was profound vascular change, there would be necrosis. He supported these statements by illustrations with numerous microscopic sections and by frequent references to published material. He also enlarged on his thesis by presentation of several case reports. He concluded by saying that both the radiation therapist and pathologist need each other in the estimation of the effects of irradiation on normal tissue and cancer.

A strong clinical orientation is needed by both: they can work together in the laboratory on problems relating to tumor growth, host immunity, and cell renewal systems. Through such combined studies, information can be rapidly accumulated that can be used in man.

In the early evening, a reception was held for all the members and guests in the Salon de Fiestas of the Hotel, the host being The Atomic Energy of Canada, Ltd. Later in the evening, the Annual Banquet was held in the Salon Independencia. Dr. James F. Nolan, President, introduced the members and wives at the Speakers' table and presented Dr. Ackerman with the Janeway Medal. Entertainment followed with presentations of native dances by local dancing teams.

The Third Scientific Session was called to order on Wednesday morning by Dr. Antolin Raventos, Davis, California, who acted as Chairman. A *Symposium on the Multidisciplinary Approach to Hodgkin's Disease* was moderated by Dr. Frederick W. George, III of Los Angeles and the panelists were Professor Sir David W. Smithers, Dr. Henry S. Kaplan of Stanford, Dr. Ralph E. Johnson of the National Cancer Institute and Dr. James Nickson of Michael Reese Hospital. These speakers presented the protocols followed in their institutions. The importance of histologic classification was emphasized in determining the management of the disease and the need for splenectomy in classifying the stage. The approach to management requires the skills of the pathologist, diagnostic radiologist, surgeon, radiotherapist and chemotherapist. A revolution in knowledge of Hodgkin's disease has taken place in recent years.

Following intermission, the morning

session continued with a presentation by Dr. J. G. Holt *et al.* on dosimetric consideration in the interstitial use of encapsulated I^{125} sources. He showed that the physical characteristics reduced considerably the radiation hazards to personnel. Dr. Vincent P. Collins *et al.* from Houston reviewed a study of anatomy and dosimetry in the treatment of carcinoma of the uterus. This study is directed to a quantitative correlation of computer dosimetry and pelvic anatomy. Dr. Frederick W. George, III *et al.* of Los Angeles expressed their views on an integrated radiotherapy system utilizing semiautomated devices with punch-card control of a cobalt 60 telecurietherapy unit. Computer assistance is used to integrate the clinical teaching and research functions. Dr. H. E. Johns of Toronto described a matrix dosimeter for high energy radiotherapy machines. A multiple dosimeter containing 49 ionization chambers covers a desired area to monitor the beam from a 35 mev. linear accelerator. Dr. Arthur Peck of New York presented interesting material collected from 40 patients regarding their emotional reactions to having cancer. Dr. Victor A. Marcial of the Puerto Rico Nuclear Center reported on the high incidence of carcinoma of the cervix with a crude rate of 36.6 cases per 100,000 women per year (1968). Dr. Gilbert H. Fletcher described his technique of extended field therapy for squamous cell carcinoma of the uterine cervix. The common iliac lymph nodes require field sizes from 17 to 20 cm. and the paraaortic lymph nodes from 28–30 cm. Dr. Julian P. Smith *et al.* from M. D. Anderson Hospital summarized their experiences of treatment of 57 patients by hypogastric artery infusion following 5,000 rads of 22 mev. betatron irradiation for advanced squamous cell carcinoma of the cervix.

An innovation to the usual program of the American Radium Society took place when Dr. Malcolm A. Bagshaw of Stanford introduced the Residents' Prize speaker Dr. Donald Goffinet of Stanford who presented his work on prolonged carotid arterial radiosensitizer infusion and radiation therapy of mouse gliomas. Prolonged intraarterial infusion of the halogenated pyrimidine bromodeoxycitidine (BrCdR) into the carotid arteries of mice in which previous intracerebral implantation of a glioma had been performed, was combined with fractionated irradiation in an attempt to develop an experimental *in vivo* model for the demonstration of radiosensitization by BrCdR and other thymidine analogues. Details of technique of irradiation and intracarotid infusion were given which resulted in prolonged survival in comparison to control groups.

During the afternoon hours, the membership and guests had the opportunity to visit the museum of anthropology for luncheon and tour followed by further sightseeing of points of interest in the City. In the early evening hours, a reception was held at Hotel El Presidente, the host being the Radium Chemical Company of New York.

The fourth and last Scientific Session of the meeting was called to order at 9:00 A. M., Thursday morning with Dr. Jerome M. Vaeth, San Francisco, California, acting as general Chairman. Dr. A. N. Arneson of St. Louis moderated the *Symposium on the Multidisciplinary Approach to Cancer of the Ovary.* The four panelists were Dr. Felix Rutledge of Houston, Dr. Lauren V. Ackerman of St. Louis, Dr. Carlos A. Perez of St. Louis and Dr. Hans-Ludvig Kottmeier of Stockholm. Dr. Rutledge emphasized the early signs of ovarian cancer and outlined ways of detection as an approach to treatment. The usual screening methods are not adequate, but should be continued until better ones are developed. Dr. Ackerman pointed out the importance of classification for help in selecting the choice method of treatment. Dr. Perez gave his indications for the role of radiotherapy in the management of these neoplasms, especially in relationship to staging. Dr. Kottmeier stated that surgery is the keystone to therapy of ovarian tumors even though a team effort is necessary in the approach to classification, radiotherapy and chemotherapy.

Following intermission, Dr. José Noriega of Mexico City related his experience since 1949 with radiotherapy of orbital tumors in a series of 249 cases in several institutions. Most of these tumors were treated in association with surgery, some with radiotherapy alone and others with chemotherapy. Forty-one patients had primary tumors and 208 secondary, either metastatic, or by direct invasion of the orbit.

Other papers presented during the latter part of the morning session were by Dr. Ronald Thompson on the management of the patient with retinoblastoma, Dr. Robert H. Sagerman on the cure of orbital rhabdomyosarcoma by radiation therapy, Dr. F. F. Gollin on combined therapy in advanced head and neck cancer, Dr. Philip M. Hatfield on long-term results of treatment of chemodectomas of the glomus jugulare and Dr. Juan V. Fayos on the therapeutic problem of metastatic neck lymphadenopathy.

Dr. M. L. Ozohan acted as substitute by reading Dr. Samuel S. Kurohara's paper on the role of radiotherapy and of surgery in the management of localized epidermoid carcinoma of the maxillary sinus.

Other presentations included those of Dr. C. A. Perez who analyzed failures and factors affecting the prognosis of malignant tumors of the tonsil in a retrospective study of 160 cases, Dr. Andrew G. Huvos who reviewed 83 patients seen over a span of 35 years with sweat gland carcinoma arising in irradiated skin, Dr. Leonard R. Prosnitz who summarized follow-up data and the value of laparotomy in the diagnosis and treatment of Hodgkin's disease and other lymphomas treated with radiation, Dr. Ralph E. Johnson who outlined integrated therapy for Ewing's sarcoma and Dr. E. Richard King *et al.* who reported on a series of 17 biopsy-proven osteogenic sarcomas of an extremity over a 15 year period which were treated with preoperative irradiation to doses of 6,000–8,000 rads followed by amputation or disarticulation of the extremity. The authors believe that the combined approach in the management of the patient with osteogenic sarcoma offers a better chance of survival and is a more rational approach than any single modality available at this time.

Following adjournment, a large number of members and guests continued their stay in Mexico by taking optional tours to Taxco and Acapulco.

The Fifty-third Annual Meeting of the American Radium Society was unusually interesting due to the excellence of the scientific program and to the unique cultural attractions of Mexico City and Mexico in general. The members and guests of the Society are indebted to its President, Dr. James F. Nolan, for his efforts in developing an interesting scientific program, to Dr. José Noriega and the members of the Arrangements Committee and to the Management of the María Isabel Sheraton Hotel for their efforts in providing many excellent facilities.

Harry Hauser, M.D.*

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* Deceased on April 27, 1971.

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edge, better ways of doing established things. He had a brilliant mind and its scope was vast. In 1949, for example, he decided that X-ray images could be sent over telephone wires so that experts in large hospitals could interpret roentgenograms transmitted from rural facilities where radiologists were scarce or of the "circuit-rider" variety. The concept was successful and he called it telognosis. With the advent of television, he updated the idea to include this medium, calling it videognosis.

He also experimented successfully with sending X rays in color in the mid 1950s; delved into gastrointestinal physiology with Dr. Harry Shay and the "Shay rat" in the 1930s; investigated such disparate subjects as the effect of tea on dental caries, special diets on the formation of bone, the etiology of Cooley's anemia, the viral etiology of cancer, and the enigmas of vanishing gallstones and the phantom nucleus pulposus. He dissected with a surgeon's skill what he considered to be our archaic medical educational system and offered suggestions for its improvement in many articles. Some 400 articles in all carried his by-line.

His major interest, however, was mammography and the early detection of breast cancer. He became convinced of the merits of breast roentgenography in the 1930s and his enthusiasm never waned. His relentless efforts over the next 40 years to persuade his colleagues of its significance contributed in large measure to the present eminence of mammography and its endorsement by the American College of Radiology. He authored 125 articles on mammography alone, as well as 2 textbooks, *Comparative Anatomy, Pathology and Roentgenology of the Breast*, with pathologist Helen Ingleby of England in 1960, and *Atlas of Mammography*, published in late 1970 by Springer-Verlag.

He pioneered in thermography in 1962, as soon as infrared-sensing devices were declassified by the military. He grasped at once the importance of such equipment to medicine and especially to early cancer detection. He was shortly considered the foremost proponent of this discipline in the country. He helped formulate the By-laws of the American Thermographic Society and became its first President (1967–68). He was a Past President of the Philadelphia Roentgen Ray Society (1965–66), and served on innumerable committees and boards of directors.

He was Director of the Division of Radiology of Albert Einstein Medical Center (formerly Jewish Hospital of Philadelphia) for 18 years (1949–66). He was also Assistant Professor of Radiology at the Graduate School of Medicine of the University of Pennsylvania (1941–68), and Professor of Research Radiology at Temple University School of Medicine (1965 until his death). He also served as consultant radiologist to small hospitals in Philadelphia and environs, among them Eagleville Sanatorium (1929–45), Skin and Cancer Hospital of Philadelphia (1935–41), the Chester County Hospital, West Chester, Pennsylvania (1938–70), Newcomb Hospital and Millville Hospital, both in New Jersey (1931–55). During World War II he served with the navy, retiring with the rank of Commander, USNR, in 1946.

A man of Jack's scientific curiosity and virtuosity obviously belonged in medicine, yet his career in this field, or in any other for that matter, was seriously in doubt during his youth. He was the eldest child of 3 boys and 5 girls born to Abraham and Dora (Starkman) Cohen, and the family was wretchedly poor. Extracurricular employment for Jack was the rule from his earliest years to supplement his father's precarious income. During his junior year at Central High School for Boys an exceptionally severe run of family misfortune compelled him to drop out and take full-time employment. He held a variety of jobs; peddled delicatessen foods his mother made from door to door, using a pushcart or a horsedrawn rig; played the violin on Friday and Saturday nights in a combo that enlivened a saloon. (Like all Jewish boys he had to master a musical instrument, and a violin was the cheapest to acquire and to be taught. He detested practicing and welcomed the day when even 25 cents for the lesson could no longer be cadged from the budget.) Only the good fortune of his receiving the Simon Muir scholarship for deserving students permitted him to return to high school and graduate with the class of 1918.

Always alert to possibilities of additional income, the young man heard of a job with a pharmacist and applied. The pharmacist turned out to be a charming young woman, Sara Eskin, who hired him on the spot and married him in 1921. With Sara's help, Jack matriculated at the University of Pennsylvania (B.S. 1922) and its School of Medicine (M.D. 1924). He interned at the Jewish Hospital (1924–25), took residency training in radiology at the University of Pennsylvania Hospital (1926–28), and opened his offices for radiologic practice in 1929. He quickly became known for the excellence of his studies and the expertness of his interpretation, and from then on the climb was all "up." Sara died in September 1968 after a union that lasted 47 years. They had no children.

Jack became an accomplished violinist, and acquired a Stradivarius of which he was justly proud. It had belonged to Catherine, Empress of Russia, and possessed extraordinary tone and beauty. He delighted in chamber music sessions with friends—usually members of the famed Philadelphia Orchestra, students of the Curtis Institute of Music, or renowned violinists in town for concerts, such as David Oistrakh (who twice tried to buy Jack's Strad, which he envied, both times without success). He loved and collected art; works of Thomas Eakins and artists of like scope covered the walls of his spacious residence in The Philadelphian Apartments, 2401 Pennsylvania Avenue. He was an omnivorous reader—of medical journals, scientific texts, philosophic treatises, and popular magazines and books. He accumulated a vast library. He was an intent listener and a penetrating conversationalist. His students found learning a joy because he made radiology interesting.

He was a Diplomate of the American Board of Radiology, certified in 1934. The perennial scholar, he extended his education to receive a D.Sc.(Med.) in 1936; and again studied to receive certification of the Society of Nuclear Medicine in 1957. He belonged to a score of scientific societies, both national and international, including the American College of Radiology, the American Roentgen Ray Society, the Radiological Society of North America, the Inter-American College of Radiology, the Society of Nuclear Medicine, the New York Academy of Sciences, the American Medical Association, and the Federation of American Societies for Experimental Biology.

He received the Alverenga Prize of the College of Physicians of Philadelphia in 1934; the Gold Medal Award of the International College of Radiology in 1937; the Man of the Year Award of the 4-8-2 Square Club of Philadelphia in 1959; the Medallion of Achievement of the Golden Slipper Square Club of Philadelphia in 1960; the Clement Cleveland Award of the American Cancer Society for outstanding work in cancer control in 1968; and the Adventure in Freedom Award for Medicine of the American Jewish Cultural Festival in 1970.

Jack remained questing, vital, and interested despite the limitations the illness of later years placed upon him. He had 15 manuscripts "in the works" on medical topics when he died, as well as a book for the laity he was writing "just for fun."

He is survived by two sisters, Sara and Esther Cohen, and a brother, Joseph.

M. B. Hermel, M.D.

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NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS OFFICERS

At the Fifty-third Annual Meeting of the American Radium Society held at the María Isabel Sheraton Hotel, México, D. F., México, March 15–18, 1971, the following officers were elected: *President*, John V. Blady, M.D., Philadelphia, Pennsylvania; *President-Elect*, Antolin Raventos, Davis, California; *1st Vice-President*, Jerome A. Urban, New York, N. Y.; *2nd Vice-President*, Morton M. Kligerman, New Haven, Conn.; *Secretary*, Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115; *Treasurer*, Victor A. Marcial, Box 20581, Rio Piedras, Puerto Rico 00928; and *Member of the Executive Committee*, James F. Nolan, M.D., Los Angeles, Calif.

The *Chairman of the Executive Committee* is Juan A. del Regato, M.D., Colorado Springs, Colorado.

The Fifty-fourth Annual Meeting of the Society will be held conjointly with the James Ewing Society and the Society of The Head and Neck Surgeons at the Boca Raton Hotel and Club, Boca Raton, Florida, May 14–19, 1972.

AMERICAN COLLEGE OF RADIOLOGY

The Forty-eighth Annual Meeting of the American College of Radiology was held at the Chase-Park Plaza Hotel, March 29–April 2, 1971.

The following officers were elected: *President*, Robert W. McConnell, M.D., Dallas, Texas; *Vice-President*, James C. Cook, M.D., Detroit, Michigan; *Secretary-Treasurer*, J. Richard Raines, M.D., Portland, Oregon; *Chairman, Board of Chancellors*, Seymour F. Ochsner, M.D., New Orleans, Louisiana; and *Vice-Chairman, Board of Chancellors*, Robert D. Moseley, Jr., M.D., Chicago, Illinois.

The following physicians have been elected as the representatives of the American College of Radiology to the Council of the Section on Radiology of the American Medical Association: Wendell G. Scott, M.D., St. Louis, Missouri; Seymour F. Ochsner, M.D., New Orleans, Louisiana; and Richard H. Chamberlain, M.D., Philadelphia, Pennsylvania.

Re-elected for the second 3 year terms to the Board of Chancellors were: Robert E. Wise, M.D., Boston, Massachusetts and J. Richard Raines, Portland, Oregon. Elected for his first term on the Board was Newton Hormick, M.D., Pittsburgh, Pennsylvania. Eugene C. Klatte, M.D., Nashville, Tennessee is the new representative of the Radiological Society of North America on the Board.

Thursday evening, April 1, 1971, there was a joint dinner with the Missouri State Chapter of the College, at which Dr. Stanley M. Wyman, President of the American College of Radiology, delivered his Presidential Address.

The Fellowship Convocation Ceremonies this year were held Wednesday, March 31, 1971 at 5:30 P.M. in the Starlight Room of the hotel. A total of 129 radiologists received the Degree of "Fellow of American College of Radiology" on the basis of "outstanding contribution and service to radiology." The Degree was formally presented to the Fellows, with Dr. Stanley M. Wyman presiding.

Gold Medals, the highest award of the College for meritorious achievement, were conferred to Dr. H. Dabney Kerr, St. Michaels, Maryland; Dr. Harold O. Peterson, Minneapolis, Minnesota; and Dr. A. Bradley Soule, Burlington, Vermont.

The ACR Teachers Conference began Thursday, April 1, 1971, at 12:30 P.M. with a Luncheon meeting, at which Dr. August G. Swanson, Director of the Department of Academic Affairs of the Association of American Medical Colleges spoke on "Trends in Undergraduate and Residency Medical Training." It continued Thursday afternoon under the Chairmanship of Milton Elkin, M.D., New York, New York; Friday morning under the Chairmanship of Thomas F. Meaney, M.D., Cleveland, Ohio; and Friday afternoon under the Chairmanship of Walter M. Whitehouse, M.D., Ann Arbor, Michigan. The Speaker at the Friday Luncheon was Thomas C. Points, M.D., Deputy Assistant Secretary for Health Services, Department of Health, Education and Welfare, whose subject was "Providing Health Care for America."

The Forty-eighth Annual Meeting of the American College of Radiology adjourned at 5:45 P.M., Friday, April 2, 1971.

The Forty-ninth Annual Meeting of the College will be held at the Americana Hotel, Bal Harbour, Florida, April 3–8, 1972.

SEMINAR ON NEWER CONCEPTS IN ABDOMINAL RADIOLOGY

The Department of Radiology at St. Peter's Hospital, Albany, New York, is presenting a 2 day Seminar on the Newer Concepts in Abdominal Radiology, on October 21 and 22, 1971.

Four basic sessions of the Seminar will be held. These are: (1) gastrointestinal, (2) biliary portal tree, (3) genitourinary or gynecologic, and (4) abdominal trauma and hemorrhage sessions.

A guest faculty of national stature has been assembled. This includes: Stanley Baum, M.D., Philadelphia, Pennsylvania; Arthur M. Clemett, M.D., Miami, Florida; Joseph T. Ferrucci, Jr., M.D., Boston, Massachusetts; Harry W. Fischer, M.D., Eloise, Michigan; Richard H. Marshak, M.D., New York, New York; James J. McCort, M.D., San Jose, California; Thomas F. Meaney, M.D., Cleveland, Ohio; Roscoe E. Miller, M.D., Indianapolis, Indiana; Henry N. Wagner, Jr., M.D., Baltimore, Maryland; Mark H. Wholey, M.D., Pittsburgh, Pennsylvania; and Robert E. Wise, M.D., Boston, Massachusetts.

Further information may be obtained from the Program Chairman, Arthur J. Wendth, Jr., M.D., Attending Radiologist, Chief, Subsection of Angiography, St. Peter's Hospital, 315 South Manning Boulevard, Albany, New York 12208.

FIRST GOLD MEDAL AWARD OF THE TEXAS RADIOLOGICAL SOCIETY

Dr. Roscoe P. O'Bannon, a member of the American Roentgen Ray Society since 1949, has been honored by the Texas Radiological Society. Dr. O'Bannon received the Society's first Gold Medal Award at its Fifty-eighth Annual Meeting conducted in Dallas, January 29–30, 1971.

Dr. W. H. Neil, President of the state group, presented the award to Dr. O'Bannon, who established his practice in Fort Worth in 1923, in recognition for his "distinguished service to radiology in Texas."

Dr. O'Bannon is a Past President and Secretary of the Texas Radiological Society and currently serves as its treasurer; he is a Fellow of the American College of Radiology since 1937; and is a distinguished member of numerous other medical and radiologic societies.

NATIONAL COUNCIL ON RADIATION PROTECTION AND MEASUREMENTS (NCRP)

Effective April 1, 1971 the address of the National Council on Radiation Protection and Measurements (NCRP) has changed *from* 4201 Connecticut Avenue, N.W., Suite 402, Washington, D. C. 20008 *to* 7910 Woodmont Avenue, Suite 1016, Washington, D. C. 20014.

The address of NCRP Publications will *not* be changed. The address for NCRP Publications remains: NCRP Publications, P.O. Box 4867, Washington, D. C. 20008.

INTERNATIONAL COMMISSION ON RADIOLOGICAL EDUCATION AND INFORMATION (I.C.R.E.)

This Commission was created by the International Society of Radiology (I.S.R.) under the Presidency of Prof. Kempo Tsukamoto.

The officers of the Commission are: *President*, Prof. Pietro Cignolini, Genova, Italy; *Vice-President*, Prof. Adolf Zuppinger, Bern, Switzerland; and *General Secretary*, Dr. James F. Ryan, Royal Prince Alfred Hospital, Camperdown 2050, Australia.

The subdivisions of the Commission are chaired as follows: *Special Committees Education*, Chairman Prof. A. Zuppinger; *Information*, Chairman Prof. P. Cignolini; *Contrast Media*, Chairman Dr. W. H. Shehadi; *Computers*, Chairman Prof. G. S. Lodwick.

The discussions of the I.C.R.E. subjects will be carried out by means of a newssheet. The first number will include the minutes of the Twelfth I.C.R.E. referring to the Commission, its regulations and provisional members, a few reports by the Chairmen regarding their programs and some partial meetings held in 1970. This is a preliminary information issue.

The task of the Chairmen is to direct the discussion. Every member of the Commission may submit problems in the form he thinks the most convenient to Prof. Pietro Cignolini, 3 Mura S. Chiara, 16128 Genova, Italia.

FIRST INTER-AMERICAN MEETING OF NEURORADIOLOGY AND THIRD BRAZILIAN MEETING OF NEURORADIOLOGY

The Inter-American Federation of Neuroradiology will hold its First Inter-American Meeting of Neuroradiology together with the Third Brazilian Meeting of Neuroradiology of the Brazilian Society of Neuroradiology in Rio de Janeiro, July 26–31, 1972.

The following topics for the Meeting have been suggested:

1. Diagnostic studies of the brain and spinal cord: (a) radiology, (b) isotopes, (c) cerebral blood flow, (d) computers, (e) ultrasound, (f) thermography, (g) others.
2. (a) Advances in basic knowledge, (b) refresher course, (c) short free papers.
3. Symposium.
4. Lectures, round table discussions.
5. Training of technicians, nurses and social assistants.

Abstracts of proffered papers should be submitted not later than January 15, 1972.

For further information, please contact: Dr. A. Tomaz Rezende, (and/or Dr. Osvaldo Bessa Fernandes, Dr. Bruno Pelizzaro) at: Caixa Postal 9031 ZC-02, Rio de Janeiro—GB—Brasil. (Tel.: 246-9402-Rio.)

The Second Inter-American Meeting of Neuroradiology will be held in the United States of America in 1976.

THIRD INTERNATIONAL CONFERENCE ON MEDICAL PHYSICS, INCLUDING MEDICAL ENGINEERING

The International Organization for Medical Physics, The Swedish National Committee for Medical Physics and The Göteborg Medical Engineering Center take pleasure in inviting scientists in the fields of medical physics and medical engineering to take part in "The Third International Conference on Medical Physics, Including Medical Engineering," which will be held in Göteborg, Sweden, July 30–August 4, 1972.

The program will include presentations of scientific papers, discussions, scientific and commercial exhibitions, and social events.

The language of the conference will be English.

Author kits will be made available through The Secretary General by October 1, 1971 in connection with the distribution of a Final Call for Papers.

For further information please write to Roland Kadefors, Secretary General, 3rd ICMP, Department of Applied Electronics, Chalmers University of Technology, 402 20 Göteborg, Sweden.

UNIVERSITY OF LONDON: MASTER OF SCIENCE IN NUCLEAR MEDICINE

A one year course leading to an M.Sc. degree in Nuclear Medicine in the Faculty of Medicine will commence in October, 1971. The course aims to provide a training for medical practitioners, or others having suitable qualifications and experience, wishing to follow careers within the Health Service, or requiring a knowledge of techniques involving the use of radioactive materials in medical research. During the course each student will be attached to an approved institution and also attend a systematic course of lectures covering physical and medical subjects.

Applications for the course should be made directly to: Dr. E. W. Emery, Department of Medical Physics, Hammersmith Hospital, Du Cane Road, London, W.12; Dr. V. R. McCready or Dr. N. G. Trott, Department of Nuclear Medicine and Department of Physics respectively, Royal Marsden Hospital, Downs Road, Sutton, Surrey; Dr. E. S. Williams, Institute of Nuclear Medicine, Middlesex Hospital School of Medicine, Nassau Street, London, W.1.

It is with deep regret that we announce the sudden death, on April 27, 1971 at his home in Cleveland, Ohio, of Dr. Harry Hauser, distinguished Associate Editor of THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE for more than 2 decades.

BOOK REVIEW

Neuroradiologic Anatomy: A Stereoscopic Atlas. By. R. S. Leighton, M.D., Chief, Radiology Service, Veterans Administration Hospital, Seattle, Wash.; and Associate Professor of Radiology, University of Washington, Seattle, Wash. Cloth. Pp. 340, with many illustrations, and 22 black and white, and color Stereo reels. Price, $55.00. Williams & Wilkins Company, 428 E. Preston Street, Baltimore, Md. 21202, 1970. GAF View-Master Lighted Stereo Viewer, $3.50.

This book is a positive addition to the understanding of the vascular anatomy of the brain. The author uses an approach similar to Bassett's *Stereoscopic Atlas of Human Anatomy*, and he incorporates the plastic injection-corrosion technique of Westberg to demonstrate the anatomy.

One chapter is devoted to the methods by which plastic vascular casts of the brain can be obtained. Dr. Leighton is ingenious in his approach to removing ventricles and cisterns from the autopsy specimen. His ability to show vessels superimposed upon cisterns or ventricles is exciting to the investigation of gross neuroanatomy.

The major portion of the book is studied in conjunction with View-master reels using a stereoscopic GAF projector.

The detail is mostly excellent, particularly the posterior fossa vasculature. The nomenclature used is coordinated with the latest terminology advocated in the neuroradiology literature.

The chapters are based upon various dissections made in multiple sections of the brain. The entire ventricular and cisternal anatomy is coordinated with the vessels which are in relationship to these structures. An inclusive index enables one to use this work as a reference for any important isolated structure.

Of 22 available reels, 16 are used for basic anatomy and all are clear and easy to follow. The other 6 reels include roentgenographic illustrations. These roentgenograms are occasionally lacking in quality and detail or are turned sideways on a given frame making it difficult to follow with the text. The external carotid artery anatomy and view of the ophthalmic artery and veins are very informative.

This work is highly recommended for the neuroanatomist and for any physician wishing to better understand the spacial relationships of various neuroanatomic structures. The finite detail makes it less appealing for the beginner. The excellent index enables one to refer to a given vessel and view it stereoscopically in several sections.

Harvey I. Wilner, M.D.

BOOKS RECEIVED

Central Nervous System Investigation with Radionuclides. Second Annual Nuclear Medicine Seminar. Compiled and edited by Albert J. Gilson, M.D., Professor of Radiology; Director, Division of Nuclear Medicine, University of Miami School of Medicine, Miami, Fla.; and William M. Smoak, III, M.D., Assistant Professor of Radiology, University of Miami School of Medicine, Miami, Fla. Cloth. Pp. 415, with many illustrations. Price, $27.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

Fundamentals of Ear Tomography. Edited by Jørgen Jensen and Hans Rovsing. Cloth. Pp. 222, with many illustrations. Price, $19.00. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

Basic Physics in Radiology. Second edition. By L. A. W. Kemp, B.Sc., Ph.D., F. Inst.P., Formerly, Chief Physicist to the London Hospital; Senior Principal Scientific Officer, Division of Radiation Science, National Physics Laboratory, Teddington, Middlesex; and R. Oliver, M.A., D.Sc., F.Inst.P., F.I.E.E., M.I. Biol., Chief Physicist in the Department of Radiation Physics of the United Oxford Hospitals; Radiation Protection Officer, University of Oxford, England. Cloth. Pp. 252, with 114 illustrations. Price, $9.00. F. A. Davis Company, 1915 Arch Street, Philadelphia, Pa. 19103, 1971.

Medical Jurisprudence. By Jon R. Walz, LL.B., Professor of Law, Northwestern University School of Law, and Lecturer in Medical Jurisprudence, Northwestern University Medical School, Chicago; and Fred E. Inbau, LL.B., LL.M., Professor of Law, Northwestern University School of Law, Chicago, Ill. Cloth. Pp. 398. Price, $10.95. The Macmillan Company, 866 Third Avenue, New York, N. Y. 10022, 1971.

Nuklearmedizin Funktionsdiagnostik. Edited by Dieter Emrica, Göttingen. Cloth. Pp. 370, with some illustrations. Price, DM 110.--. Georg Thieme Verlag, Stuttgart. In Canada and U.S.A., Intercontinental Medical Book Corporation, New York, N. Y. 10016, 1971.

CLINICAL UROGRAPHY. An Atlas and Textbook of Roentgenologic Diagnosis. In Three Volumes. Third edition. By John L. Emmett, M.D., M.S. (Urology), Emeritus Consultant, Section of Urology, Mayo Clinic; Emeritus Professor of Urology, Mayo Graduate School of Medicine (University of Minnesota), Rochester, Minn.; and David M. Witten, M.D., M.S. (Radiology), Formerly, Consultant, Section of Diagnostic Roentgenology, Mayo Clinic; Assistant Professor of Radiology, Mayo Graduate School of Medicine (University of Minnesota), Rochester, Minn. Cloth. Pp. 2,101 with 2,621 illustrations. Price, $84.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1971.

RADIOGRAPHY OF INFANTS AND CHILDREN. Second edition. By Donald B. Darling, M.D., Associate Professor of Radiology and Pediatrics, Tufts University School of Medicine; Pediatric Radiologist-in-Chief, Tufts–New England Medical Center; Assistant Clinical Professor of Radiology, Boston University School of Medicine; Clinical Instructor in Radiologic Technology, Northwestern University. Foreword by John Caffey, M.D., Professor Emeritus of Radiology, College of Physicians and Surgeons, Columbia University; Consultant Radiologist, Columbia Presbyterian Medical Center, New York; Visiting Professor of Radiology and Pediatrics, School of Medicine, University of Pittsburgh. Cloth. Pp. 211, with 301 figures. Price, $28.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

USE AND INTERPRETATION OF THE LUNG SCAN. By Fred S. Mishkin, M.D., Associate Professor of Radiology; Director of Nuclear Medicine, Indiana University Medical Center; Consultant and Chief of Nuclear Medicine, West 10th Street V.A. Hospital, Indianapolis, Ind.; Associate in Radiology, Nuclear Medicine, Marion County General Hospital; and Richard E. Brashear, M.D., Associate Professor of Medicine, Indiana University Medical Center, Indianapolis, Ind. Cloth. Pp. 121, with many figures. Price, $12.25. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

EXERCISES IN DIAGNOSTIC RADIOLOGY: 2; The Abdomen. By Lucy Frank Squire, M.D., Lecturer on Radiology, Harvard Medical School; Visiting Radiologist, Massachusetts General Hospital, Boston, Mass.; William M. Colaiace, M.D., Lecturer in Medical Science, Brown University; Radiologist, Roger Williams Hospital, Providence, Rhode Island; and Natalie Strutynsky, M.D., Assistant Professor, Radiology, New York Medical College, New York, N. Y. Paper. Pp. 88, with 122 illustrations. Price, $4.95. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1971.

THE RADIOLOGIC CLINICS OF NORTH AMERICA. Symposium on Selected Techniques in Radiology. April, 1971. Volume IX. Number 1. Guest Editor, Murray L. Janower, M.D. Cloth. Pp. 160, with some illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1971.

FORTSCHRITTE IM STRAHLENSCHUTZ: Synopsis über 10 Jahre Forschung; Strahlenschutz in Forschung und Praxis—Band X. Edited by Heribert Braun, Friedrich Heuck, Hans-Adolf Ladner, Otfried Messerschmidt, and Ludwig Rausch. Paper. Pp. 259, with some figures. Price, DM 48.-. Georg Thieme Verlag, Stuttgart. In Canada and U.S.A., Intercontinental Medical Book Corporation, New York, N. Y. 10016, 1970.

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SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Sheraton Hotel, Boston, Mass., September 28–October 1, 1971.

American Radium Society
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Boca Raton Hotel and Club, Boca Raton, Fla., May 14–19, 1972. Conjoint meeting with the James Ewing Society and the American Society for Head and Neck Surgery.

Radiological Society of North America
Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 28–December 3, 1971.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Bar Harbour, Florida, Americana Hotel, April 3–8, 1972.

Section on Radiology, American Medical Association
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga., 30322. Annual meeting: Atlantic City, N. J., June 20–24, 1971.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Bal Harbour, Fla., June 7–11, 1971, Americana Hotel; Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel; Washington, D.C., June 5–9, 1972, Washington-Hilton Hotel; and Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel.

Written examinations are scheduled in June of each year in 13 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined. The written examinations this year will be held on June 19, 1971.

Deadline for filing applications for any examination in 1972 is September 30, 1971.

American Association of Physicists in Medicine
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

American Society of Therapeutic Radiologists
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104

American Institute of Ultrasound in Medicine
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

American Society of Neuroradiology
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

Thirteenth International Congress of Radiology
Meeting: Madrid, Spain, Oct. 13–19, 1973.

Tenth Inter-American Congress of Radiology
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16-22, 1971.

Inter-American College of Radiology
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

Second Congress of the European Association of Radiology.
President, Professor Dr. J. R. von Ronnen, State University of Leiden, The Netherlands.
Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, The Netherlands. Congress Meeting: Amsterdam, The Netherlands, June 14–18, 1971.

First Asian and Oceanian Congress of Radiology
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia. Meeting: Melbourne, Australia, Nov. 22–26, 1971.

Alabama Chapter of ACR
Secretary, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

American Thermographic Society
Secretary-Treasurer, Dr. Irwin M. Freundlich, Department of Radiology, Thomas Jefferson University Hospital, Philadelphia, Penn. 19107. Annual Meeting: Sheraton-Deauville Hotel, Atlantic City, N. J., June 20, 1971.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. Wilma C. Diner, Univ. of Arkansas Medical Center, Little Rock, Ark. 72201. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Elliott C. Lasser, University Hospital of San Diego County, San Diego, Calif. 92103. Annual Meeting: Durham, N. C., May 13–15, 1971, with the Duke University and University of North Carolina Radiology Departments serving, as co-hosts.

Atlanta Radiological Society
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Lucien F. Burke, Maj, MC, Box 44, 97th Gen. Hosp., APO New York 09757. Meets quarterly. Fall meeting Oct. 1971, in Berlin, Germany.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Kenneth B. Robinson, 301 E

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

75th St., Apt. 11-A, New York, N.Y. 10021. Meets first Thursday of each month, October through June.

Buffalo Radiological Society

Secretary, Dr. Glen M. Ebersole, 405 Spring St., Jamestown, N.Y. 14701. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiological Society, California Chapter of ACR

Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

Catawba Valley Radiological Society

Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society

Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society

Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society

Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society

Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR

Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society

Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society

Secretary-Treasurer, Dr. Fred H. Dunn, 5940 Forest Park Rd., Suite 101, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR

Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society

Secretary-Treasurer, Dr. John C. Osmer, 20103 Lake Chabot Rd., Castro Valley, Calif. 94546. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society

Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR

Secretary, Dr. Wm. F. Lindsey, 1215 Hodges Dr., Tallahassee, Fla. 32303. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society

Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR

Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society

Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society

Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society

Secretary-Treasurer, Dr. George J. Meyer, Holy Cross Hospital, Ft. Lauderdale, Fla., 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists

Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Ghim L. Yeoh, Prof. Center Bldg., 1481 S. King St., Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 P.M.

Health Physics Society

Secretary. John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Waldorf Astoria Hotel, New York City, July 11–15, 1971.

Houston Radiological Society

Secretary, Dr. Milton L. Wagner, 6621 Fannin, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR

Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR

Secretary, Dr. Jack L. Melamed, Grant Hospital of Chicago, 551 Grant Pl., Chicago, Ill. 60614. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR

Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR

Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society

Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society

Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society

Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society

Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society

Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR

Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society, Chapter of ACR

Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 p.m. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 p.m.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 p.m., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 p.m.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101. Biennial Symposium at the Holiday Inn, Bozeman, Mont., July 28–30, 1971.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. N. Patrick Kenney, 3618 S. 114th Ave., Omaha, Nebr. 68144. Meets third Wednesday of each month at 6 p.m. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89502.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 p.m.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary-Treasurer, Dr. Samuel H. Madell, 1 E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 p.m. Annual Spring Conference: Waldorf Astoria Hotel, New York. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. John W. Morris, III., Department of Radiology, Halifax District Hospital, Daytona Beach, Fla. 32015. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Barbara Chick, Glens Falls Hospital, Glens Falls, N.Y. 12801. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiological Society
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Edward I. Miller, 301 Newport Blvd., Newport Beach, Calif. 92660. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Gerald L. Warnock, 11699 N. E. Glisan St., Portland, Ore. 97220. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 p.m., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook

Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07206. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver. Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 19–21, 1971.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, 20 Bridge Rd., Kentfield, Calif. 94904. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Raymond L. Schwinn, 480 Monterey Ave., Los Gatos, Calif. 95030. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Sheraton Hotel, Boston, Mass., September 26–27, 1971.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Haakon O. Haugan, 716 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Sixteenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 28–30, 1972.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Lawrence R. Nickell, Maury County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Marriott Hotel, Houston, Tex., March 23–25, 1972.

The Fleichner Society
Secretary-Treasurer, Eric N. C. Milne, M.B., Medical Sciences Bldg., University of Toronto, Ontario, Canada.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. R. Newell Ford, St. Mark Hospital, 803 North 2nd West, Salt Lake City, Utah 84103.

Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR

Secretary, Dr. Edward A. Kupic, Mary Fletcher Hosp., Burlington, Vt. 05401.

Virginia Chapter of ACR

Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501.

Washington, D. C., Chapter of ACR

Secretary-Treasurer, Dr. William E. Sheely, 1231 Kingston Ave., Alexandria, Va. 22302.

Washington State Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Leland L. Burnett, 5765 S. Eddy St., Seattle, Wash. 98118. Meets quarterly.

West Virginia Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society

Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

Wyoming Radiological Society, Chapter of ACR

Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología

Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.

Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología

Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador

Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala

Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.

Coahuila No. 35, México 7, D.F., México.

Secretary-General, Dr. Octavio Toussaint Goribar. Meets first Monday of each month.

Sociedad Radiológica Panameña

Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico

Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 p.m. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quèbec

Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology

Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.

Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Edmonton and District Radiological Society

Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists

Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting, Oxford, England, July 2–3, 1971.

Faculty of Radiologists, Royal College of Surgeons in Ireland

Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)

Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists

Honorary Secretary, Dr. F. Robert MacDonald, *Associate Honorary Secretary*, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting.

Montreal Radiological Study Club

Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association

Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie

Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

Toronto Radiological Society

Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

College of Radiologists of Australasia

Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología

Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia

Secretary, Dr. Víctor A. Añaños, Instituto de Radiología, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 p.m. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia

Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología

Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología

Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia

Secretary, Dr. Armando Rocha Amoêdo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia

Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología

Secretary, Dr. Ricardo Cortez, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raúl Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Julio Ormeño del Pino, Instituto de Radiología "Cayetano Heredia," Hospital Arzobispo Loayza, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de la Plata
Secretary. Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Miguel A. Sica, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Mario A. Moya, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Octavio Machado, Colegio de Médico del Dístrito Federal, Plaza de Bellas Artes, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Rontgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Permanent Secretary, Clément Fauré, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France

Société Française d'Electroradiologie Médicale and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Rontgengesellschaft
President, Professor Dr. med. Hanno Poppe, Radiologische Univ.-Klinik, Gossler Str. 10, 34 Göttingen, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. C. B. A. J. Puijlaert, Prof. Dondersstraat 73, Tilburg, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C.-E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. D. José Bonnati, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Schweizerische Gesellschaft für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Philippine College of Radiology
Secretary-Treasurer, Dr. Edmundo V. Villacorta, Box 1284, Commercial Center, Makati, Rizal D-708, Philippines. Meets fortnightly on Fridays. Annual Meeting: Jan. 1972.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan, Ibadan, Nigeria.

South African International radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ROENTGEN DIAGNOSIS

ABDOMEN

SCHULENBURG, C. A. R. (Pretoria, South Africa.) Operative cholangiography in acute cholecystitis. *Med. Proc.*, Sept., 1970, *16*, 318–321.

The author performs operative cholangiography in all cases of cholecystectomy.

All patients with acute cholecystitis are operated on as soon as their condition allows, usually within the first 3 days after admission.

Of 1,100 consecutive cholecystectomies, 94 were in cases of acute cholecystitis and in each case operative cholangiography was attempted, in 92 successfully. Essentially no complications were attributed to the roentgenographic studies. Unsuspected common duct stones were found in a few cases.

The author feels that it is not only advisable but essential that common duct stones be sought at operation for acute cholecystitis, as in the case of "cold" operations.—*Donald R. Hawes, M.D.*

SCHULENBURG, C. A. R. (Pretoria, South Africa.) Anomalies of the biliary tract as demonstrated by operative cholangiography. *Med. Proc.*, Oct., 1970, *16*, 351–354.

In a series of 1,093 operative cholangiographies anatomic anomalies of the biliary tract were demonstrated in 230 cases (21 per cent).

These were classified as follows:

I. Anomalies of cystic duct (13.7 per cent), consisting of variations in length, variations in site of confluence of major ducts, and variations in side or site of entry of the cystic duct into the hepatic duct.
II. Anomalies of hepatic duct (2.9 per cent), consisting of anomalous ducts (accessory hepatic duct), or variations in level of junction of right and left hepatic ducts.
III. Anomalies of the common bile duct, consisting of 1 cyst (duplication), and 1 hamartoma.
IV. Variations in position of duodenal papilla (5.6 per cent).
V. Anomalies of gallbladder, consisting of 1 instance of agenesis and double gallbladder.—*Lois Cowan Collins, M.D.*

REDMAN, HELEN C., REUTER, STEWART R., and JOSEPH, RAMON R. Roentgenographic evaluation of patients with suspected obstructive jaundice. *Surg., Gynec. & Obst.*, Dec., 1970, *131*, 1100–1104. (From: Departments of Radiology and Internal Medicine, Wayne County General Hospital, Eloise, Mich.; and University of Michigan, Ann Arbor, Mich.)

Routine roentgenographic and laboratory examinations do not distinguish drug-induced jaundice from obstructive jaundice and such patients may undergo an unnecessary exploratory laparotomy.

Combined angiography and percutaneous transhepatic cholangiography were used in 15 patients with biochemical findings suggestive of obstructive jaundice. The presence or absence of biliary obstruction was correctly determined in 13 of the 15 patients and in retrospect the other 2 cases could have been correctly diagnosed on the basis of the roentgenograms.

The presence or absence of obstruction was correctly determined by percutaneous transhepatic cholangiography in 13 patients. The 2 falsely negative cases were due to failure to enter a duct in one case, and limitation of the stricture to the left bile duct in the other.

The cause of the obstruction was diagnosed correctly by angiography in 7 of the 11 patients with obstruction and retrospectively in all 11. Percutaneous transhepatic cholangiography led to a correct diagnosis in 7 of the 11 patients and in retrospect it could have been made in 9 of the 11.

The value of these two diagnostic procedures in distinguishing hepatocellular jaundice, particularly drug-induced, from obstructive jaundice lies in the fact that the only other method of reliable differentiation is laparotomy.—*Lois Cowan Collins, M.D.*

GENITOURINARY SYSTEM

EL MAIRY, ASAAD B., and CHEHATA, OMAR A. Clinical evaluation of new methods of pyelography. *Kasr-El-Aini J. Surg.*, July, 1970, *11*, 247–256. (From: Minshawy Tanta Hospital, Tanta, Egypt.)

During the past decade 2 major advances in intravenous pyelographic techniques have emerged. These are: the double or multiple dose method; and the drip infusion pyelography. The modern radiologist may use one or both of these methods routinely, or he may use them to supplement the standard single dose technique under certain circumstances.

Radiology is a developing art. Methods used many years ago may not be advisable in the seventies. There are a number of reasons for eliminating abdominal compression and dehydration which in past years have been employed to enhance the pyelographic pattern. Dehydration is useless if renal blood flow is impaired, and it can precipitate oliguria or anuria as well as renal failure in cases of hyperparathyroidism, myelomatosis, and underlying conditions associated with dehydration. Abdominal compression can provoke vasovagal attacks and is contraindicated where there is abdominal trauma, masses, colic, etc.

The authors evaluated 60 cases with the double or multiple dose method and the drip infusion technique. Normal hydration was used and compression eliminated. They feel that the results were far superior to the standard technique and saved several patients from needless operations when the routine pyelography showed a nonfunctioning kidney. Drip infusion pyelography seemed indicated in cases with ischemic kidneys as in chronic pyelonephritis, in impairment of renal blood flow as in senile arteriosclerosis, in elderly dehydrated cases, and in borderline cases of liver failure. Extravascular infusion can be used in very obese patients as well as in children. Visualization of the kidneys was often obtained even when the blood urea nitrogen was over 60 mg. per cent.—*Arch H. Hall, M.D.*

Šnobl, O., Švorc, J., and Dibelková, M. Die Markschwammniere im Kindesalter. (Medullary sponge kidney in childhood.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1970, *113*, 644–650. (Address: Doc. MUDr. O. Šnobl CSc., Röntgenabteilung der I. und II. Univ.-Kinderklinik, Praha, 2,/CSSR, Sokolská 2.)

Medullary sponge kidney is a congenital cystic disease involving renal pyramids exclusively. In early life, the condition tends to remain asymptomatic and can only be suspected clinically when a secondary infection with uremia develops. Occasionally, however, the disease may also be recognized in earlier life.

Six pertinent cases have been observed in children with an age range of 6 to 14 years, including 5 girls and 1 boy. In a different series, a marked male preponderance has been observed. The diagnosis on hospital admission varied from nephrocalcinosis with pyelonephritis to renal acidosis, tubular insufficiency and postglomerulonephritis status.

Characteristic roentgen findings in conventional abdominal views comprise minute mulberry-type medullary calcifications converging on the papillae of the renal pyramids. Intravenous urograms will disclose contrast medium in the pyramids with a fan-shaped appearance and ring formations occasionally resembling a bouquet-of-flowers. Enlarged papillae may change the shape of the minor calyces which can appear broader and ovoid with an irregular contour.

Characteristic clinical signs include polyuria and polydipsia. A secondary pyelonephritis may lead to a metabolic acidosis and renal colic. These variable clinical signs can obscure the underlying medullary sponge kidney, especially when complications develop.

In the differential diagnosis, one has to exclude renal tuberculosis with calcifications, nephrocalcinosis with tubular acidosis, nephrolithiasis, renal papillary necrosis and calyceal diverticula. Staining of the pyramids also occurs in pyelotubular reflux. In this condition, however, there is absence of minute cyst formations which are so characteristic of medullary sponge kidney.—*Ernest Kraft, M.D.*

Marquis, James R., and Khazem, Bernard. Sickle-cell disease: renal roentgenographic changes in children. *Radiology*, Jan., 1971, *98*, 47–52. (From: Departments of Radiology and Pediatrics, Children's Hospital of Newark, United Hospitals Medical Center, Newark, N. J.)

The kidneys, in sickle-cell disease, have been well documented clinically and pathologically, but there have been relatively few reports in the radiologic literature. The most frequently described major renal changes are filling defects and papillary necrosis. The authors were able to find only 1 article describing renal roentgen changes in children with sickle-cell anemia.

The authors' paper is based on 25 patients under 16 years of age (17 females and 8 males) who underwent excretory urography. Five males and 14 females were proved S-S; 3 males and 3 females were S-A.

Poor concentration was found in only 3 patients, possibly due to larger doses of contrast material. Twelve patients had calyceal blunting, apparently unrelated to infection in view of negative urine cultures and absence of genitourinary symptoms. Kidney enlargement greater than 1 S.D. was found in 7 patients. There was no roentgen evidence of renal papillary necrosis or filling defects in the renal pelvis.

The basic pathologic process in sickle-cell disease is intravascular stasis and congestion, thrombosis, and fibrosis occurring in the kidney and elsewhere.

The most common roentgen change was blunting of the minor calyces and was referred to as "sickle-cell caliectasis" to distinguish it from bacterial inflammatory pyelonephritis or obstructive caliectasis. Ten patients had bilateral and 2 patients had unilateral calyceal blunting of varying severity. This finding is most likely due to congestion, necrosis, and fibrosis.

The significance of renal enlargement is not clear, but may be a manifestation of congestion. The enlargement may be striking and can mimic polycystic disease or an infiltrative process such as leukemia.

No change in kidney size or caliectasis was demonstrated in 3 of 4 patients, who had serial urographic studies over a period of time ranging from 1 to 7 years.

The authors could find no correlation between the roentgen changes and the clinical course or age of the patient.—*J. G. Hutka, M.D.*

Johnson, A. J., and Kotsis, H. N. Refluxing ureteral stump with pseudocyst formation: a complication of renal homotransplantation. *J. Urol.*, Nov., 1970, *104*, 668–669. (From: Division of Urology, Henry Ford Hospital, Detroit, Mich.)

This is a case report. Following bilateral nephrectomy with high ureteral ligation and subsequent uneventful renal transplantation, the patient returned with complaints of incomplete bladder emptying and frequency, and had a palpable low abdominal midline mass.

Cinecystourethrography revealed vesicoureteral reflux into the right ureteral stump.

Drained via suprapubic catheter, the mass contained 2,500 cc. of clear urine.

Surgery later demonstrated the mass to be a urine-containing pseudocyst, which communicated with the right ureter.—*Hugh F. Gardiner, M.D.*

Williams, Arthur V., Derrick, Fletcher C., Jr., and Hargest, Thomas S. A disposable radiopaque grid for percutaneous renal biopsy. *J. Urol.*, Nov., 1970, *104*, 646–648. (From: Departments of Medicine [Renal Section], Urology and Surgery [Engineering Development Section], Medical University of South Carolina, Charleston, S. C.)

Percutaneous renal biopsy is a frequently employed procedure in the evaluation of obscure renal disease. It is fraught with certain hazards, the most frequent of which is hemorrhage (hematuria, perirenal hematoma, etc.).

The authors feel that this is usually secondary to improper placement of the biopsy needle. They present a technique using a disposable radiopaque grid to increase accuracy in renal localization for biopsy. They cite a series of 141 percutaneous renal biopsies and 12 cyst aspirations in which there were no complications. They do not employ fluoroscopic control. —*Robert J. Gubler, M.D.*

Skeletal System

Serre, H., Simon, L., Sany, J., and Blotman, F. L'ostéonécrose primitive du genou chez le sujet agé: à propos de 18 cas. (Primary osteonecrosis of the knee in the elderly: report of 18 cases.) *Presse méd.*, Nov., 1970, *14*, 2119–2122. (From: Clinique de Rhumatologie et Clinique d'Orthopédie, Clinique Saint-Eloi, C.H.I., F.-34-Montpellier, France.)

Less well known than the more common osteochondritis dissecans of young persons is the rarer primary knee osteonecrosis of the elderly. Eighteen cases in persons over 50 years of age were unilateral, and 12 were in women. Unlike juvenile osteochondritis, there is usually a sudden onset of severe knee pain, with progression, and without antecedent acute trauma. Aseptic effusion is common; there is no joint locking. One-half of the authors' cases showed genu varum; one-half of the patients were obese; 2 had associated vascular abnormalities; and there was 1 each with gout, diabetes, and old syphilis.

Although radiography is the basis of diagnosis, the symptoms may precede the roentgen findings by months. Air-contrast arthrography or Sr^{85} scanning may reveal early changes.

The earliest plain roentgenographic finding is a mild flattening or irregularity of the medial femoral condyle in the lateral view. One such case showed a strongly positive Sr^{85} uptake. Progression leads to appearance of a small flattened break of the distal condyle margin or a small zone surrounded by sclerosis. Later, there is typically a semilunar break of the condylar margin, surrounded by an irregular sclerotic band, and limited below by a thin sequestrum, usually not detached. In contrast to juvenile osteonecrosis, these lesions are more medial, being quite a distance from the intercondylar notch, and are larger and less sharply defined.

In the absence of successful treatment, the natural history often leads to an increasing medial tibiofemoral arthrosis. Few favorable outcomes were obtained by available medical or surgical means in the current series.

(This reviewer strongly recommends for additional reading "Spontaneous Osteonecrosis of the Knee," Ahlbäck *et al.*, *Arthritis and Rheumatism*, 1968, *11* 705–733, which contains a superb series of successive roentgenographic illustrations of this same entity.)—*Alan E. Oestreich, M.D.*

Numaguchi, Yuji. Osteitis condensans ilii, including its resolution. *Radiology*, Jan., 1971, *98*, 1–8. (From: Department of Radiology, Atomic Bomb Casualty Commission, Hiroshima, Japan.)

Forty-eight cases of osteitis condensans ilii, all in Japanese females, are presented and the results compared with previous reports. The mean age was 35 years. Ninety-two per cent had been pregnant. The prevalence was 1.16 per cent, a lower figure than the prevalence reported from Europe.

Roentgenographically, the condition consists of varying degrees of osteosclerosis lateral to the sacroiliac joints. This sclerosis may be unilateral (12.5 per cent) or bilateral; if bilateral, asymmetric involvement was seen in about half of the cases. While opinions differ regarding sacral involvement, in nearly all cases in this series a narrow band of sclerosis was noted on the sacral side of the sacroiliac joint as demonstrated in the 45° oblique projection. In 16 of the 19 patients followed greater than 8 years, partial or complete resolution of the sclerotic densities resulted. This probably accounts for the rarity of the condition in older patients.

While 26 patients initially presented low back pain, most (17) had other associated roentgenographic abnormalities, such as scoliosis or narrow intervertebral disk spaces, to explain the pain. No correlation was found between the degree of pain and the extent of bone involvement by the sclerotic process.

Etiologically, no correlation was noted between osteitis condensans ilii, and urinary tract infection, moderate obesity, or history of trauma. No correlation was noted between the severity of the roentgenographic involvement and the number of pregnancies or length of labor.

Differential diagnosis includes Marie-Strümpell's spondylitis, tuberculosis, nonspecific or syphilitic osteoarthritis, Paget's disease, and Garre's osteomyelitis.—*I. Whitfield Carhart, M.D.*

Jardon, O. Max, Burney, Dwight W., and Fink, R. L. Hypophosphatasia in an adult. *J. Bone & Joint Surg.*, Oct., 1970, *52-A*, 1477–1484. (From: Department of Orthopaedics, University of Nebraska and Affiliated Hospitals, Omaha, Neb.)

Only 10 cases of hypophosphatasia in an adult have been reported in the literature. The disease is characterized by low serum alkaline phosphatase, skeletal abnormalities and presence of phosphoethanolamine in the urine and serum.

The roentgenograms may show fractures, pseudofractures, subperiosteal new bone formation and femoral bowing.—*Richard P. Taylor, M.D.*

Diamond, Liebe S. A family study of spondyloepiphyseal dysplasia. *J. Bone & Joint Surg.*, Dec., 1970, *52-A*, 1587–1594. (From: J. L. Kernan Hospital, Baltimore, Md. 21207.)

A study of 6 generations of a family with a genetic disease best described as a mixture of multiple epiphyseal dysplasia and spondyloepiphyseal dysplasia is presented. Detailed information is available for 4 generations of 44 members, 31 of whom reveal some stigmata of the condition.

The following findings are noted: (1) coxa vara; (2) changes in the hip resembling all stages of avascular necrosis; (3) right thoracic and thoracolumbar scoliosis of some degree; (4) vertebral body abnormalities either those of multiple epiphyseal dysplasia tarda showing irregular surfaces of the vertebrae with disk intrusions into the body and anterior wedging of isolated vertebra, or of spondyloepiphyseal dysplasia tarda with flat vertebral bodies, and narrow disks with irregular vertebral plates; (5) severe degenerative arthritis of the hips; (6) severe to moderate changes with wide shallow acetabula with irregular walls; (7) stunted stature but not true dwarfism; and (8) normal hands, feet and knees.—*S. A. Kaufman, M.D.*

Sholkoff, Stephen D., Glickman, Morton G., and Steinbach, Howard L. Roentgenology of Reiter's syndrome. *Radiology*, Dec., 1970, *97*, 497–503. (From: Department of Radiology and the Rheumatic Disease Group, Department of Medicine, University of California School of Medicine, San Francisco, Calif.)

The roentgenograms of 55 patients with well documented Reiter's syndrome, during the years 1966 through 1968, were reviewed and the frequency and distribution of roentgenographic changes of Reiter's syndrome are discussed.

There was a tendency toward asymmetric arthritic involvement, with erosions and effusions the most frequent findings, and with periostitis noted less often. The most common sites of involvement were the heel, toe, and sacroiliac joints.

In the authors' series, roentgenograms in 10 out of 55 patients with Reiter's syndrome (18 per cent) demonstrated no abnormality.

The distribution of the arthritic roentgenographic findings would often permit a presumptive diagnosis in the absence of a classical clinical history.—*William L Graham, M.D.*

Gordon, Robert B. Arthrography of the ankle joint: experience in one hundred seven studies. *J. Bone & Joint Surg.*, Dec., 1970, *52-A*, 1623–1631. (Address: 2299 Post Street, San Francisco, Calif. 94115.)

Arthrographies of 107 ankles were performed.

The technique of ankle arthrography is discussed. It is simple, safe and accurate.

It is particularly reliable in determining the site and extent of soft tissue injury.—*Richard P. Taylor, M.D.*

Blood and Lymph System

Stelzig, H.-H., Meyer, W. W., and Siebert, G. Über die Verkalkung der atherosklerotischen Beete in den Becken- und Extremitätenarterien: Mikroradiographische und histologische Untersuchungen. (Calcification of the atherosclerotic beds in pelvic and extremity arteries: Microradiographic and histologic examinations.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1970, *113*, 614–627. (Address: Dr. med. H.-H. Stelzig, Institut für Klinische Strahlenkunde, 65 Mainz, Langebeckstrasse 1, Germany.)

In spite of voluminous literature, the morphogenesis and histotopography of calcification of atherosclerotic foci has not been clarified. Conventional roentgen techniques, even with fine grain film emulsions, have proved inadequate for solving micromorphologic problems. Application of microradiography, on the other hand, has greatly aided in the study of vascular calcifications. A combination of the macroscopic and microradiographic demonstration of calcifications enables a specific histologic and histochemical analysis of the arteries.

For the specific studies, the iliac, femoral and popliteal arteries of 43 necropsy tissues were obtained. The vessels were opened longitudinally and subjected to a modified v. Kossa reaction. Microradiographic examinations were performed on 75 arterial segments, 8 cm. long, and on 22 transverse or longitudinal cuts, 0.8 to 1.0 mm. thick.

The mineralization of the atherosclerotic plaques presents various shapes. Calcification of the bed of the intima plaques varies from microscopic dust to granular formations and large shells. The mineralization represents a progressive lesion with increasing size of the calcium particles.

Dust-like and granular calcifications can also be found in the diffusely thickened intima adjacent to the plaques. The media in this area usually are thin. The inner elastic membrane below the plaques is markedly fragmented. In the necrotic atheroma, the necrotic parts of the bed remain free from calcium.

Presence of calcium dust around large plaques suggests that new calcium particles appear continuously in the bed. Thus, the plaques become increasingly larger. Microradiographically, one can follow the progressive closure of the gaps between the atherosclerotic foci. The localization of the calcium also changes from one focus to the next.

The conditions influencing the mineralization and demineralization of the atherosclerotic foci and their surroundings are discussed in detail.—*Ernest Kraft, M.D.*

LEEDS, NORMAN E., and GOLDBERG, HERBERT T. Lenticulostriate artery abnormalities: value of direct serial magnification. *Radiology*, Nov., 1970, *97*, 377–383. (From: Departments of Radiology, Montefiore Hospital and Medical Center, the Albert Einstein College of Medicine, Bronx, N. Y., and the Albert Einstein Medical Center, Philadelphia, Pa.; and the Department of Radiology, Philadelphia General Hospital, Philadelphia, Pa.)

Lenticulostriate arteries are usually not well seen on routine cerebral arteriograms because of their small size. Anteroposterior and lateral direct serial magnification delineates these arteries better, and, therefore, in a limited series of patients, abnormalities are recognized which otherwise would be unnoticed.

The authors successfully demonstrated minor irregularities of the arteries as well as small microaneurysms in hypertension, actual bleeding sites in patients with intracerebral hemorrhages, mild arterial displacements, areas of avascularity, and small areas of tumor infiltration. These findings are often not seen in routine cerebral arteriography, and, therefore, aid in the clinical evaluation of the patient.

The technique to obtain detailed visualization of the lenticulostriate arteries by serial magnification is described and involves the use of a fine focal spot (0.3 mm.), a tube-object distance of 15 to 17 inches, and an object-film distance of 23 to 25 inches. The object is taken as a point midway between the outer canthus of the eye and the tragus. With a horizontally fixed changer the tube should be angled no more than 10°, or a special base may be constructed to permit angulation of the changer to maintain its position perpendicular to the tube.—*Carl H. Weidenmier, Jr., M.D.*

EHRINGER, H., FISCHER, M., LECHNER, K., and MAYRHOFER, E. Thrombolytic therapy of non-acute arterial occlusions. *German Med. Monthly*, Oct., 1970, *15*, 621–631. (Address: I. Medizinische Universitätsklinik, Spitalgasse 23, A-1090 Wien, Austria.)

An investigation has again confirmed the principle that lysis of arterial occlusions is possible *in vivo*. An analysis of the results by age and site of the occlusion has shown that streptokinase therapy is remarkably successful in the treatment of occlusions of the iliac, femoral and popliteal arteries of up to 6 weeks' duration.

The history appeared to be important in assessing the likelihood of success of streptokinase therapy. In all cases in which successful lysis was obtained, the patient was able to pinpoint to the very day the onset of severe ischemic symptoms occurring without prior warning even though pre-existing chronic symptoms had been present. Angiographic findings before and after therapy left no doubt that generalized vascular disease was present.

In this study of 35 patients it was found that after successful thromboembolic therapy, the peripheral blood flow was of the same order as after successful surgical vascular reconstruction. Improvement in blood flow has been maintained for up to 2½ years with continuous anticoagulant therapy.

Radiologists should be aware of the different forms of therapy available for this condition.—*Arch H. Hall, M.D.*

HEUCK, F., DEININGER, H.-K., and VANSELOW, K. Ergebnisse der Röntgen Kine-Densitometrie der Organdurchblutung. (Results of the roentgen cinedensitometry of blood flow in organs.) *Röntgenblätter*, Dec., 1970, *23*, 593–598. (Address: Prof. Dr. F. Heuck, Zentralröntgeninstitut des Katharinenhospitals, 7000 Stuttgart, Kriegsbergstrasse 60, Germany.)

For functional analysis of an organ, testing of specific blood flow may prove very helpful. The procedure is described in detail. A very small dose of contrast material is injected into an artery and rapid serial exposures are made simultaneously; also, the arterial pressure curve and frequency of exposures are registered. The progression of the bolus of contrast material and the diameter of the artery are measured. Thus, the flow-time volume can be determined with tagged contrast material as an indicator. Cineroentgenograms will then give the highest resolution for electronic densitometry.

The comparable cinedensitometry was first determined in the normal and abnormal circulation of the brain and the kidney. The median value of the blood-flow volume of the internal carotid artery lies between 5 and 6 ml./sec. With increased intracranial pressure these values will decrease. The blood-flow volume of the renal artery depends on the size and weight of the kidney. In the average healthy kidney, it amounts to 600–700 ml./min. With increased blood pressure, the blood-flow volume increases. Lower volumes are encountered in renal hyperplasia, nephrosclerosis and renal infarcts.

The densitometry curve simultaneously shows flow phenomena of the renal artery. It also can register the blood flow in different zones of the renal parenchyma.—*Ernest Kraft, M.D.*

RADIATION THERAPY

Henk, J. M., Kunkler, P. B., Shah, N. K., Smith, C. W., Sutherland, W. H., and Wassif, S. B. Hyperbaric oxygen in radiotherapy of head and neck carcinoma: interim report of a controlled clinical trial. *Clin. Radiol.*, July, 1970, *21*, 223–231. (From: South Wales Radiotherapy Centre, Velindre Hospital, Whitchurch, Cardiff, Wales.)

The value of hyperbaric oxygen in radiotherapy has remained controversial although 13 years have elapsed since its first use was reported by Churchill-Davidson *et al.* The authors attempt to evaluate the role of hyperbaric oxygen as an adjuvant to radiotherapy in a prospective controlled clinical trial initiated in September 1964, following preliminary experience of the technique extending over the preceding 12 months. Tumors of the head and neck were utilized and a regimen of 10 fractions in 3 weeks, allocating each patient at random to treatment of either air or oxygen after prescription of the tumor dose. Identical beam direction techniques were used in air and oxygen treatments.

The criteria for admission to the trial were as follows: (1) radical external beam radiotherapy was considered as the treatment most appropriate, excluding small tumors in the mouth usually treated by implant, and laryngeal carcinomata confined to mobile vocal cords; (2) the patient was under the age of 75; (3) the diagnosis of carcinoma was histologically proven before treatment; (4) no previous radiotherapy or surgery, definitive in character, had been carried out; (5) the patient was considered medically fit to undergo treatment in the hyperbaric chamber; (6) no metastases were present other than those in the cervical lymph nodes.

The trials stratified into 4 groups: Group 1, nasal and oral carcinoma; Group 2, laryngeal carcinoma; Group 3, laryngopharyngeal carcinoma; Group 4, carcinoma in cervical lymph nodes when the primary site could not be found, or a primary carcinoma not in the above groups.

All patients received a tumor dose between 3,500 and 4,500 rads from a 4 mev. linear accelerator in 10 equal fractions over 3 weeks; *i.e.*, treatment 3 times a week on Mondays, Wednesdays and Fridays.

Patients allocated to oxygen were treated at a pressure of 3 atmospheres absolute in a Vickers oxygen tank.

Higher survival rates and significantly higher local tumor control rates have so far been observed in the oxygen compared with the air series, with no difference in complications.

The authors conclude that under the conditions of this trial, high-pressure oxygen is an effective adjuvant to radiotherapy.—*Luther W. Brady, M.D.*

Murphy, G., Moore, R. H., and Kenny, G. M. Current results from primary and secondary treatment of renal cell carcinoma. *J. Urol.*, 1970, *104*, 523–527. (From: Department of Urology, Roswell Park Memorial Institute, New York State Department of Health, State University of New York, Buffalo, N. Y.)

Survival rates following primary or secondary treatment of renal cell carcinoma are thought to be in need of current re-evaluation. Questions relative to postoperative radiotherapy in improving survival rates for these patients have been raised. Some investigators have pointed out that more than half of the patients without initial metastases survive more than 5 years following nephrectomy with or without irradiation as a primary treatment, while others advocate more aggressive surgical intervention and in selected instances preoperative radiation therapy.

The authors report their results of primary and secondary treatment of patients with renal cell carcinoma at the Roswell Park Memorial Institute.

The results of 192 patients undergoing primary or secondary therapy for renal cell carcinoma between 1960 and 1968 were reviewed. Eighteen patients were lost to follow-up. Cases were divided on the basis of presence or absence of known initial metastases. Initial metastases were present in 79.7 per cent of the patients, yet the average survival rate exceeded 2 years.

The 5, 10 and 15 year survival rates for patients without initial metastases were 45, 25 and 25 per cent, respectively.

A 50.6 per cent 1 year survival rate was achieved in 87 patients receiving secondary therapy (irradiation, chemotherapy and androgens) for secondary metastases.

A significant difference was noted as a result of secondary treatment for metastatic renal cell carcinoma for as long as 5 years.—*Luther W. Brady, M.D.*

Coucourde, F., and Maestro, A. The management of recurrences of malignant tumors of the uterine cervix by 42 MeV betatron. *Radiol. clin. et biol.*, 1970, 39, 407–418.

(From: Institute of Radiology and Radiotherapy, University of Pavia, Pavia, Italy.)

Radiotherapy alone or combined with surgery can provide satisfactory results in the treatment of malignancies of the uterine cervix. Recurrence of these tumors may occur after varying periods of time. By the term "recurrence" it should be understood that this means the "reappearance of clinical symptoms of the tumor after a period of subjective or objective symptomatic silence, following a primary therapeutic intervention performed with the intention of being radical." On the other hand, the surgical intervention or the radiologic treatment, when incomplete or inadequate with objective clinical findings being present, is more properly described as "persistent disease."

The authors describe their experience since August 1965 in the Institute of Radiology of the University of Pavia using a 42 mev. Siemens betatron which emits, for medical purposes, a 42 mev. photon beam and a high energy electron beam from 5 to 42 mev. In the interval from August 1965 through December 1967, 33 patients with recurrent tumors of the uterine cervix were treated. The authors describe the radiation techniques employed and the results achieved. The sites of recurrence varied from the cervix to the parametrial areas to the pelvis. The time of appearance of recurrence varied from 1 to 10 years following primary treatment, with 1 patient having a recurrence 13 years after treatment and 2 after 24 years.

In general the early results have been satisfactory with the pelvic and parametrial infiltrates noticeably being reduced and in 2 patients who suffered unilaterally from marked edema of the lower extremity, a complete disappearance of the edema was observed. Best results were obtained in patients with vaginal or cervical recurrences, either strictly localized or involving the base of the parametria. Complete tumor regression at the vault and a re-epithelization of the cervix could be observed. Even in parametrial and pelvic recurrences good results were obtained.

The authors found no observable relationship between the outcome of treatment and the clinical features of the recurrent disease. Twelve patients are still alive 10 to 36 months after the end of the radiation therapy, 7 having had vaginal or cervical recurrences with or without initial involvement of the parametrium, 2 having primary parametrial recurrence and 3 having recurrent pelvic disease.

The conclusion is drawn that hard photon radiation beams of this quality are useful in treating recurrent cervix carcinoma, giving generally satisfactory results. It is emphasized that radiation therapy with ultrahard photon radiations must be used in patients with recurrences of the cervix, even when the treatment has to be strictly palliative.—*Luther W. Brady, M.D.*

BLOOM, H. J. G., RICHARDSON, W. W., and FIELD, J. R. Host resistance and survival in carcinoma of breast: a study of 104 cases of medullary carcinoma in a series of 1,411 cases of breast cancer followed for 20 years. *Brit. M. J.*, July, 1970, *3*, 181–188. (Address: H. J. G. Bloom, M.D., Consultant Radiotherapist, Royal Marsden Hospital and Institute of Cancer Research, London S.W.3, England.)

For many years the study of human cancer was focused chiefly on the inherent characteristics and manifestations of the tumor itself. There has been increasing interest recently in the response of the host and in the concept of natural resistance to malignant disease.

The authors report their experience with a special type of mammary carcinoma, generally of high-grade malignancy, carrying a remarkably good prognosis—so-called "medullary carcinoma of the breast with lymphoid infiltration." They suggest that the increased lymphoid tissue seen in these tumors is concerned with cell-mediated and humoral immunologic reactions and reflects a strong host-defence mechanism related to the remarkably high survival rates following radical treatment. Since the evidence for host resistance to malignant disease is based largely on animal data, the opportunity to study a group of patients followed for 20 years, in whom this type of defence reaction appeared to exist, was thought by the authors to be of considerable clinical interest.

Among 1,411 cases of breast cancer there were 104 with medullary carcinoma (7.4 per cent), for which corrected 5 to 20 year survival rates were calculated. After 20 years, 74 per cent of the cases with operable medullary tumors were alive, compared with 14 per cent of cases with similar stage non-medullary cancer. In the presence of histologically proved axillary metastases, the 20 year survival rate was 61 per cent for medullary cases, compared with only 13 per cent for other types of breast cancer.

In 30 cases of medullary cancer in which the axilla was free, the corrected 20 year survival rate was 95 per cent following a combination of radical operation and radiotherapy. No evidence could be found that axillary dissection or postoperative irradiation is harmful to women with operable highly malignant breast cancer in whom a well-marked host resistance is thought to be present. A combination of radical mastectomy and postoperative irradiation appears to be the most effective treatment for such cases.

The authors conclude from their study that the present grounds for rejecting a radical approach to the treatment of breast cancer, based on current immunologic considerations, are regarded as being quite inadequate.—*Luther W. Brady, M.D.*

KAISER, R. Endocrine mechanisms offering protection against endometrial and mammary carcinoma. *German Med. Monthly*, June, 1970, *6*, 309–315. (From: Department of Obstetrics and Gynecology, University of Munich, Munich, Germany.)

Patients with mammary and endometrial carcinoma often show characteristic endocrine, constitutional and fertility patterns. The author reviews the literature regarding endocrine factors involved in the prevention or etiology of mammary and endometrial carcinoma.

Women with endometrial and mammary carcinoma often have a history of reproductive inefficiency as evidenced by anovulatory cycles, infertility or a lower than average number of children. In spite of the high estrogen concentrations noted during pregnancy and the luteal phase of the menstrual cycle, these times represent spells of progestational dominance. At this point, target organs show diminished proliferative activity. Synthetic progestogens and combined estrogen-progestogen preparations used for the inhibition of ovulation have the same effect on target organs. Each phase of the progestogen dominance resembles castration, except for the associated depletion in steroid hormones and atrophic changes. Diminished proliferative activity is seen during lactation when secretory activity of the mammary parenchyma occurs in association with ovarian inactivity and an absence of endometrial stimulation.

Breaks from proliferative activity offer protection against mammary and endometrial carcinoma. Ovarian dysfunction, associated with long spells of proliferative activity and hyperestrogenism, cause an excess of cell proliferation and turnover between puberty and a frequently delayed menopause. This factor, together with a genetic predisposition to carcinoma, plays an important role in the etiology of carcinoma. The author points out the relevance noted that patients with endometrial carcinoma are often android and obese, showing signs of excess adrenal and islet-cell activity with diminution in thyroid function.

The author suggests the generous use of progestogens as a method of prophylaxis in patients with anovulatory cycles. He also suggests that it might be extended to puberty in view of the increasing incidence of breast carcinoma in younger women. Inhibition of ovulation is suitable in mature and premenopausal women. It would seem appropriate to seek to abolish any pathologic prolongation of proliferative activity. He points out, however, that there may be objections to properly indicated and controlled estrogen therapy.—*Luther W. Brady, M.D.*

Gill, W., Long, W., Fraser, J., and Lee, P. Cryosurgery for neoplasia. *Brit. J. Surg.*, July, 1970, *57*, 494–502. (From: Department of Clinical Surgery, University of Edinburgh, Edinburgh, Scotland.)

Destruction of living tissue by cold was first described by Dr. James Arnott of Middlesex Hospital in 1851, where fungating tumors were treated with a salt-ice mixture with observed reduction in size, pain, odor, discharge and hemorrhage. Little advance was made in cryotherapy during the next century, although Pusey in 1907 used solid sticks of carbon dioxide to freeze skin lesions.

The evolution of cryosurgical techniques is now well documented and stems from engineering advances in 1961 by Cooper. For the first time it was possible to freeze, in a controlled fashion, lesions in deep-seated tissues such as the brain.

The authors report their experience over a period of 3 years in the clinical professorial unit of Edinburgh University with 99 patients with various forms of neoplasia treated by cryosurgery. The tumors included benign and malignant tumors of the prostate, benign and malignant tumors of the skin, malignant tumors of the rectum, maxilla, chest wall and gluteal region.

The authors' experience in dealing with these neoplasms confirmed their opinion that the majority of skin tumors, benign and malignant, are best treated by cryosurgery, which has advantages over radiation therapy and conventional excision techniques. Cryotherapeutic results on large tumors such as sarcomas and rectal carcinomas are less satisfactory for technical reasons.

Cryoprostatectomy on a highly selected group of patients produced encouraging results, although the authors still regard this method of treatment as in its infancy and requiring further assessment.—*Luther W. Brady, M.D.*

RADIOISOTOPES

Hayek, Alberto, Chapman, Earle M., and Crawford, John D. Long-term results of treatment of thyrotoxicosis in children and adolescents with radioactive iodine. *New England J. Med.*, Oct., 1970, *283*, 949–953. (From: Departments of Pediatrics and Medicine, Harvard Medical School, the Children's and Medical Services, Massachusetts General Hospital, and the Shriners Burns Institute, Boston, Mass.)

The authors relate their experience in 30 children and adolescents (ages 8–18 years) treated with radioiodine for thyrotoxicosis. These patients (28 females and 2 males) were observed for a mean of 9.2 years after receiving an average of 6.6 mc I^{131} (28 patients) or I^{130} (2 patients).

In 25 patients prompt remission was obtained with a single administration of radioiodine. Eight of these patients (26 per cent) developed permanent hypothyroidism. Three of the 5 patients who received 2 doses of radioiodine have become hypothyroid; this was thought to be dose related. No deaths and no evidence of cancer or leukemia were seen in these 30 patients.

The authors conclude that the information available to date in children with thyrotoxicosis treated with radioiodine gives assurance that this form of therapy is "related" in safety to surgery and the antithyroid drugs. They caution the use of radioiodine in children less than 5 years of age and in children with very large goiters.—*A. Everette James, Jr., M.D.*

Samuels, Larry D., Grosfeld, Jay L., and Kartha, Mukund. Liver scans after primary treatment of tumors in children. *Surg., Gynec. & Obst.*, Nov., 1970, *131*, 958–964. (From: Departments of Pathology and Surgery, Division of Pediatric Surgery, Children's Hospital and the Ohio State University; and Division of Radiation Therapy, Department of Radiology, the Ohio State University, Columbus, Ohio.)

The effect of ionizing radiation upon the liver may be assessed by accumulation of radiopharmaceuticals in both hepatic and reticuloendothelial cells. Livers of children appear sensitive by this method to absorbed doses of 2,000–3,000 rads delivered in 2 weeks to adjacent organs.

Scanning of the liver was performed in the 5 patients on a rectilinear scanner (5 inch NaI crystal) operated at a speed of 100–200 cm. per minute. Either Tc^{99m} sulfur colloid (0.5–1.5 mc) or I^{131} rose bengal (50–100 μc) was the radiopharmaceutical employed. Four of the 5 patients received dactinomycin, a potentiating drug, prior to radiation therapy.

The effects of irradiation upon the liver is evidenced on scans as areas of diminished radioactivity and relative increase in size of other portions of the liver. Parenchymal and reticuloendothelial cells appear equally sensitive to irradiation.

To properly analyze these effects a baseline liver scan should be obtained prior to therapy and serially afterwards.—*A. Everette James, Jr., M.D.*

Monetti, N., Abbati, A., and Bellanova, B. Contributo della scintigrafia con ^{85}Sr eseguita mediante whole body scanner nello studio delle metastasi scheletriche da ca. mammario. (Contribution of scintigraphy with Sr^{85} performed by whole body scanner in the study of skeletal metastases from mammary ca.) *Ann. radiol. diag.*, Sept., 1969, *42*, 495–508. (From: Istituto di Radiologia e Terapia Fisica dell'Ospedale Maggiore di Bologna, Italy.)

Strontium 85 whole body scanning was performed in 25 patients who presented with cancer of the breast. The majority of the cases had a mastectomy, while a few cases were inoperable. The scanning was performed 2, 5, and in a few cases 15 days after the administration of the radionuclide, in the amount of 1 μc/kg. body weight, and always less than 1,000 microcuries.

The more significant findings were noted in the scanning performed 5 days after the introduction of the strontium 85.

On the basis of the data obtained, detailed roentgenographic investigation was performed of the areas which showed an increased calcio-metabolic activity.

From an analysis of the cases studied it is felt that strontium 85 whole body scanning can demonstrate latent metastatic foci, and that this study should be performed before surgery and/or radiotherapy in order to plan the most appropriate treatment.—*A. F. Govoni, M.D.*

Krishnamurthy, G. T., Winston, Martin A., Weiss, Edward R., and Blahd, William H. False-positive pulmonary photoscans obtained with a dual-probe detector system. *J. Nuclear Med.*, Dec., 1970, *11*, 719–722. (Address: G. T. Krishnamurthy, Nuclear Medicine Service, B-114, Rm. 228, Veterans Administration Center, Los Angeles, Calif. 90073.)

This article is of considerable clinical importance to all who perform scintiphotographic studies of the lung, particularly when dual-probe scanning systems are used for this purpose.

The authors clearly demonstrate that in performing lateral pulmonary photoscanning, the dependent lung does not expand normally, causing alterations in its appearance on the photoscan. They found that there was no significant disparity in the appearance of the anterior or posterior pulmonary photoscan images, when these were obtained with the patient lying in either the supine or prone position.

They attribute the observed artifacts in the lateral photoscans to several factors, which include upper displacement of the diaphragm by the dependent abdominal viscera, compression of the dependent lung by the lower thoracic cage, and compression of the lingula and segments of the upper and lower lobes by the cardiac structure (particularly when the patient is in the left lateral scanning position).

Four figures are shown which include anatomic sketches of the lung to demonstrate the marked change in its appearance when scanned in the dependent as opposed to nondependent positions.—*Merle K. Loken, M.D.*

Lorber, Steven A., Gold, Fred M., Maglione, Anthony A., and Rubenfeld, Sidney. ^{69m}Zn-chloride—a new scanning agent: a study of its dosimetry and biological fate. *J. Nuclear Med.*, Dec., 1970, *11*, 699–703. (Address: Steven A. Lorber, New York University Medical Center, School of Medicine, 550 First Avenue, New York, N. Y. 10016.)

The authors present their experience using a new radioisotope, zinc 69m, for scintiphotographic studies. Zinc 69m has a 13.8 hour physical half-life and emits a .438 mev. gamma ray suitable for detection by most instrumentation used in nuclear medicine. They administered 7 mc per kg. of this radioisotope into 25 dogs and up to 33 mc per kg. into 18 rabbits and studied its rate of accumulation and elimination from various body organs. From the data obtained they determined the relative concentrations and radiation doses to these organs.

Their data show that the highest concentration of zinc 69m chloride was found in the liver with lesser amounts in the prostate, pancreas and kidney. The radiation absorbed doses to these organs were felt by the authors to be within an acceptable range for use in clinical nuclear medicine.

Scintiphotograms of the prostate gland show localization of the radioisotope within this structure. However, the resolution appears marginal so that it seems doubtful that this scinctiphotographic procedure will yield useful morphologic information on this organ.—*Merle K. Loken, M.D.*

MAHLER, DELMAR J., SCOTT, ARTHUR F., WALSH, JOHN R., and HAYNIE, GORDON. A study of trace metals in fingernails and hair using neutron activation analysis. *J. Nuclear Med.*, Dec., 1970, *11*, 739–742. (From: Veterans Administration Hospital and Reed College, Portland, Oregon.)

The authors report their experience in the analysis of hair and fingernails from 18 dialysis patients and 12 uremics whose creatinine clearance was less than 5 and 5–12 ml. per min. respectively. Their study included 13 control individuals.

The specimens were thoroughly cleansed using deionized water and alcohol and placed into polyethylene vials which were in turn placed in a nuclear reactor (TRIGA MARK 1) for a period of 2 hours at a flux of 2×10^{12} neutrons per cm.^{2} per second. The gamma spectra of all samples were obtained using either a sodium iodide or germanium detector with 400 and 4,096 channel analyzers, respectively.

Their results show considerable variability, but despite this, manganese and zinc appeared to be higher in the fingernails of dialysis patients than in controls, whereas sodium, copper and gold were lower in these patients. Results of the uremic patients studied agreed more closely with the results of the dialysis group than they did with the control group except for manganese and copper. The differences between trace metal levels in the hair of controls and patient groups were negligible, except for manganese in uremics, which appeared lower than either in the control or dialysis group.

Their data also show that trace metal content varies considerably with geographic locations from which the subjects originate, thus reflecting environmental differences in soil and water minerals.—*Merle K. Loken, M.D.*

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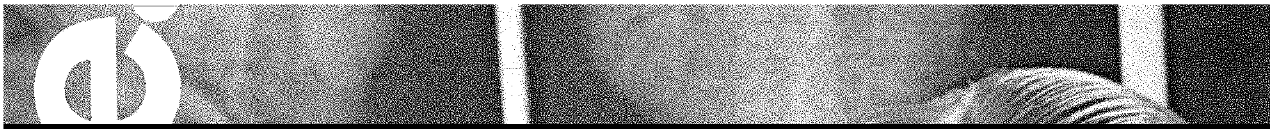

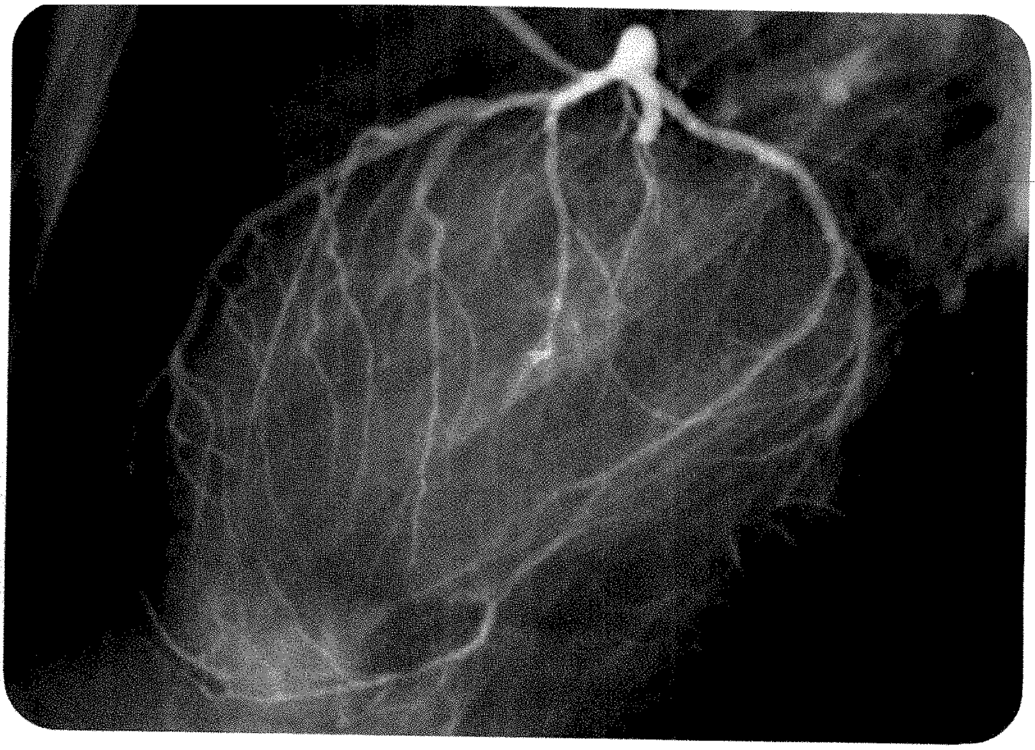

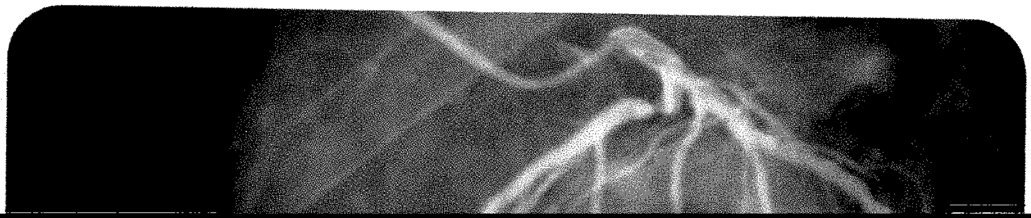

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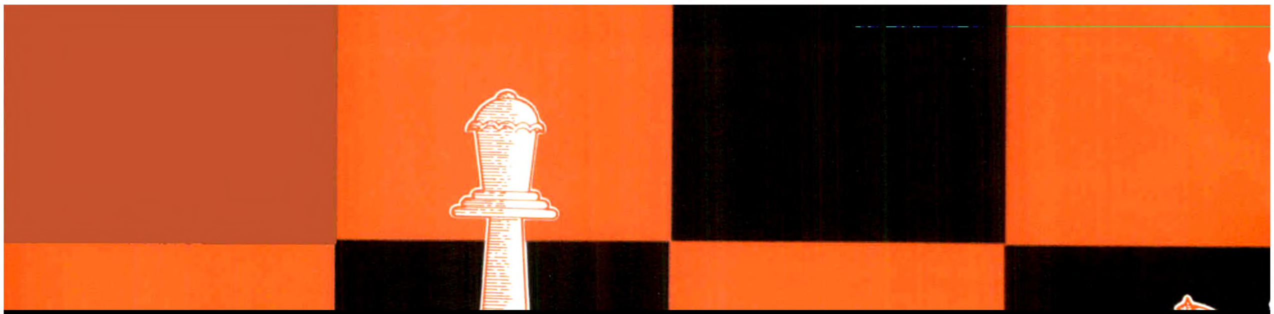

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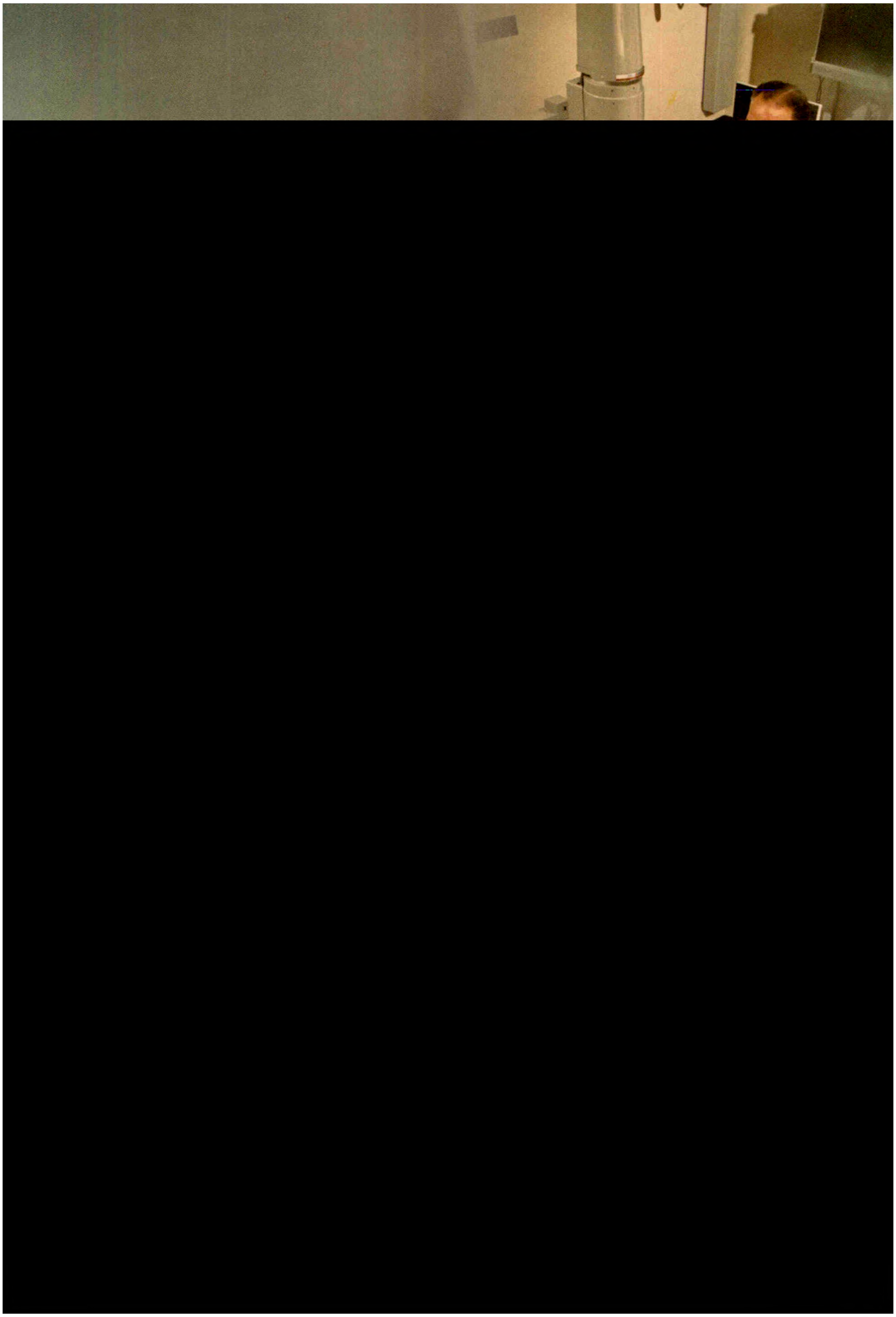

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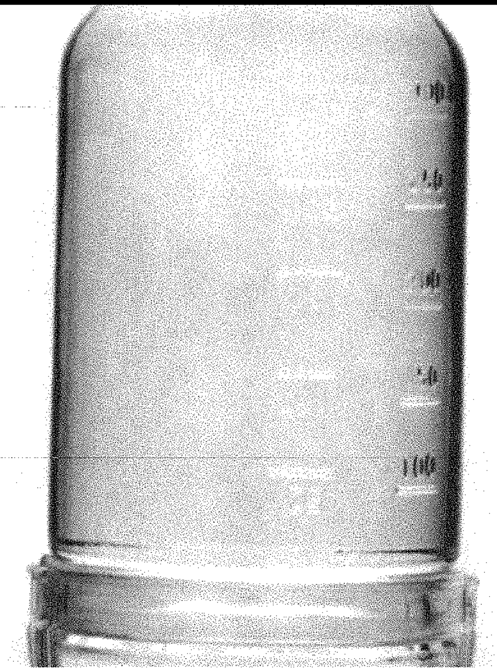

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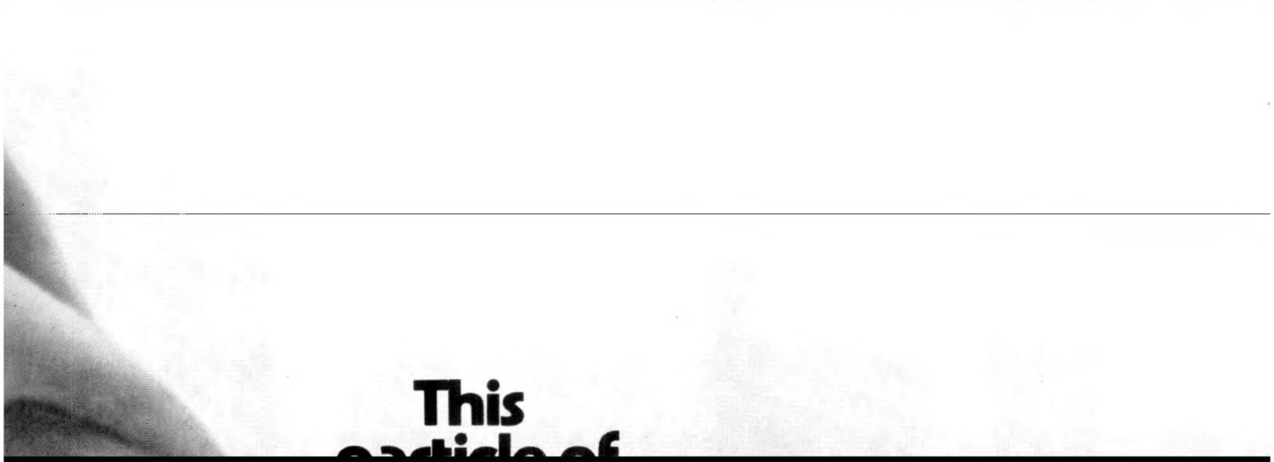

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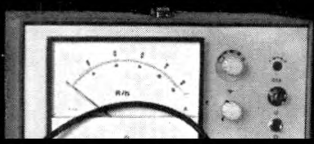

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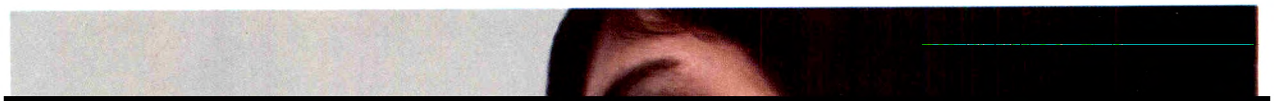

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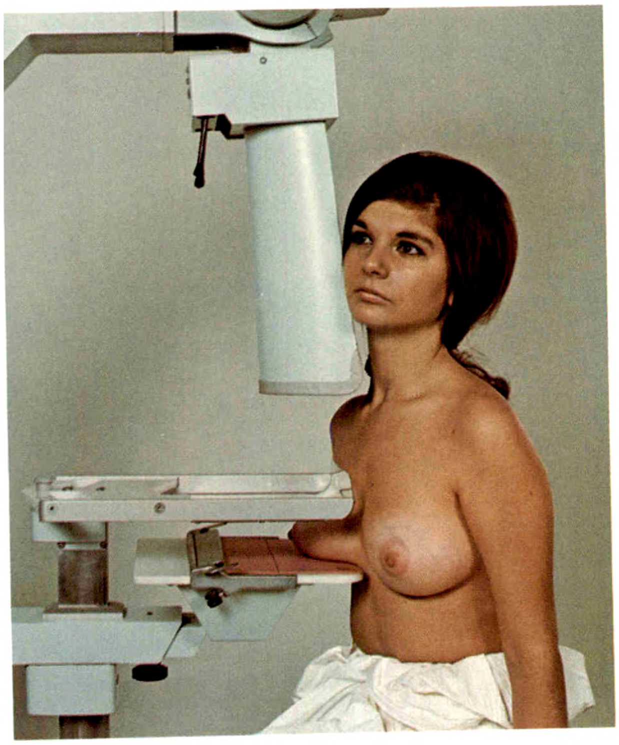

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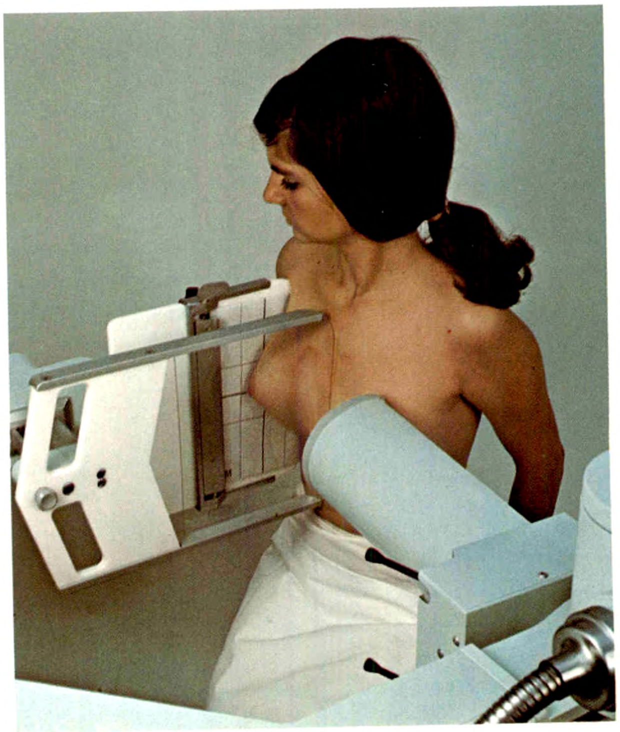

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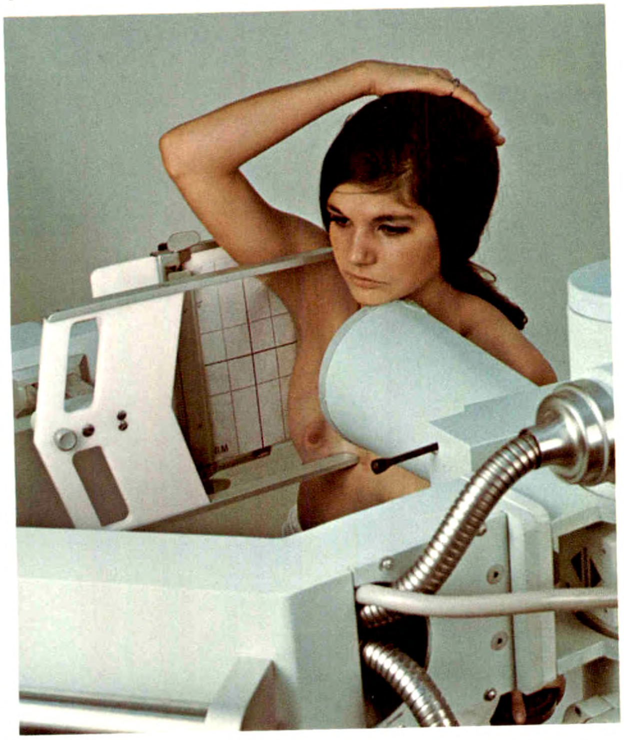

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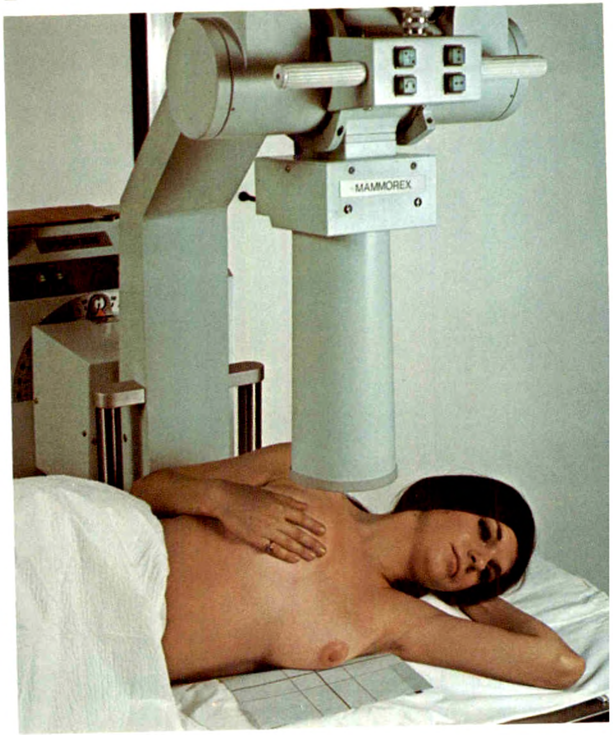

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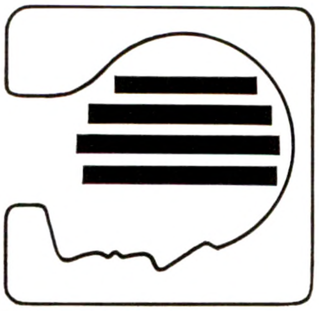

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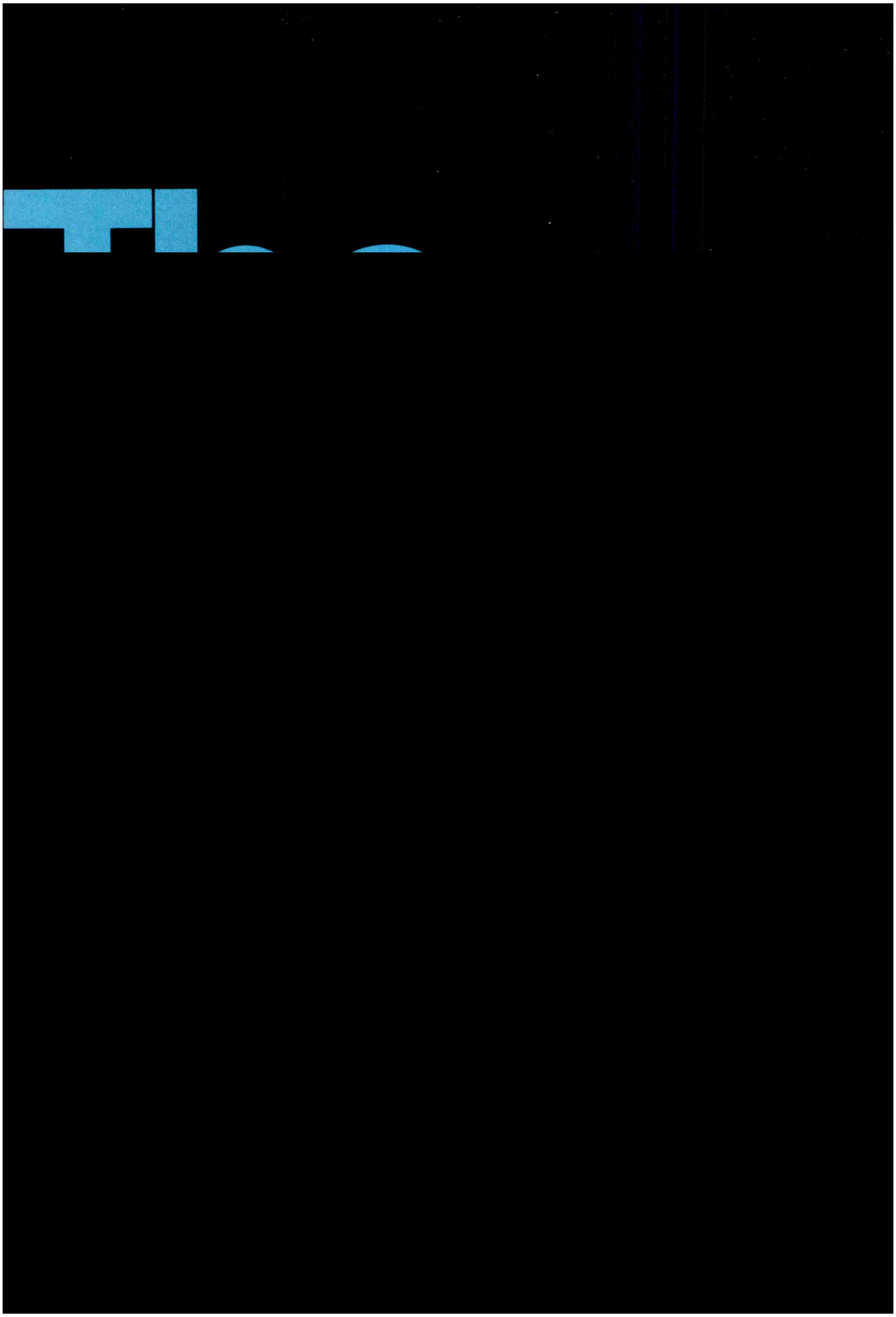

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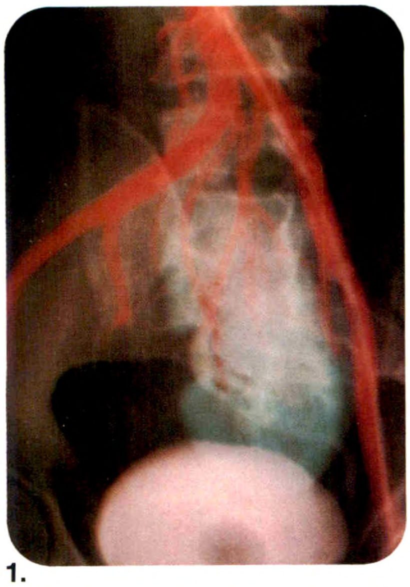
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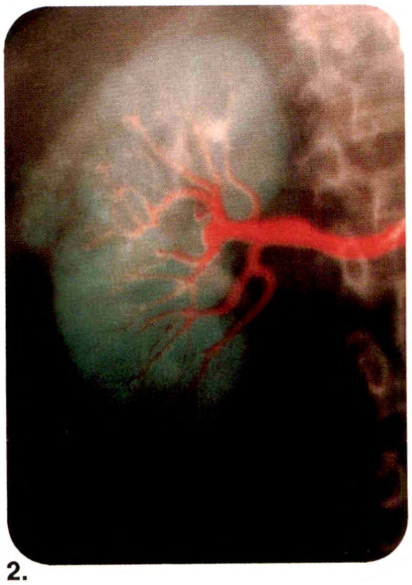
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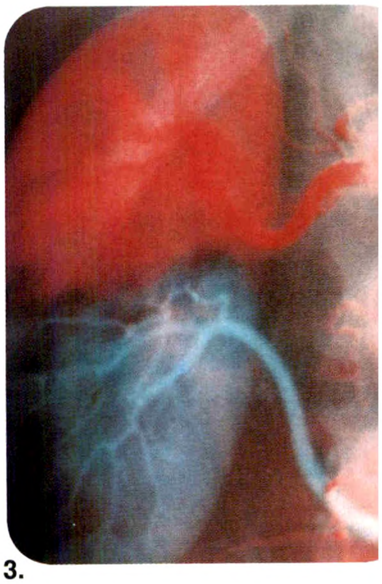
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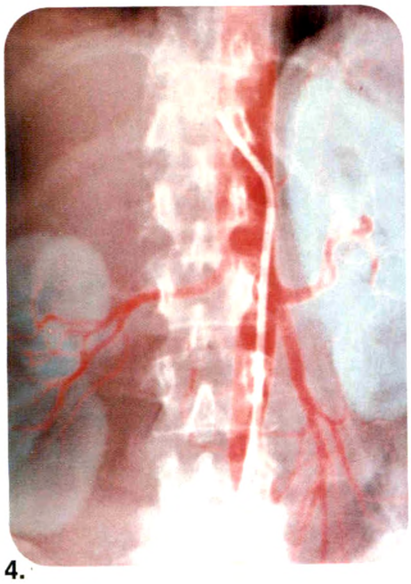
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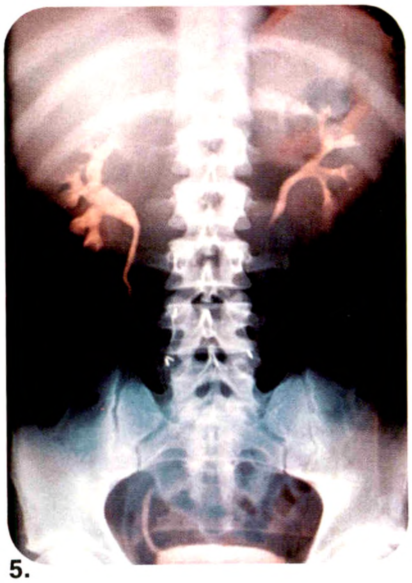
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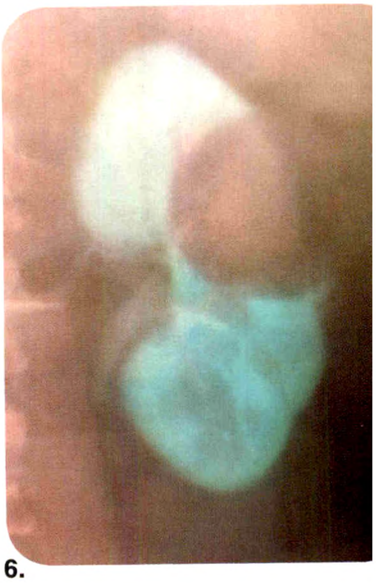
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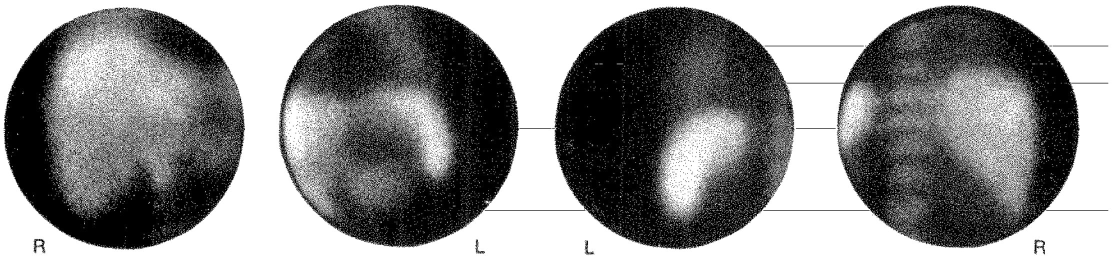

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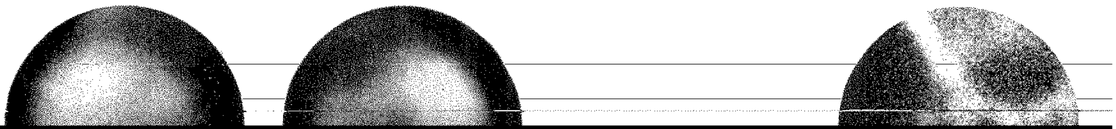

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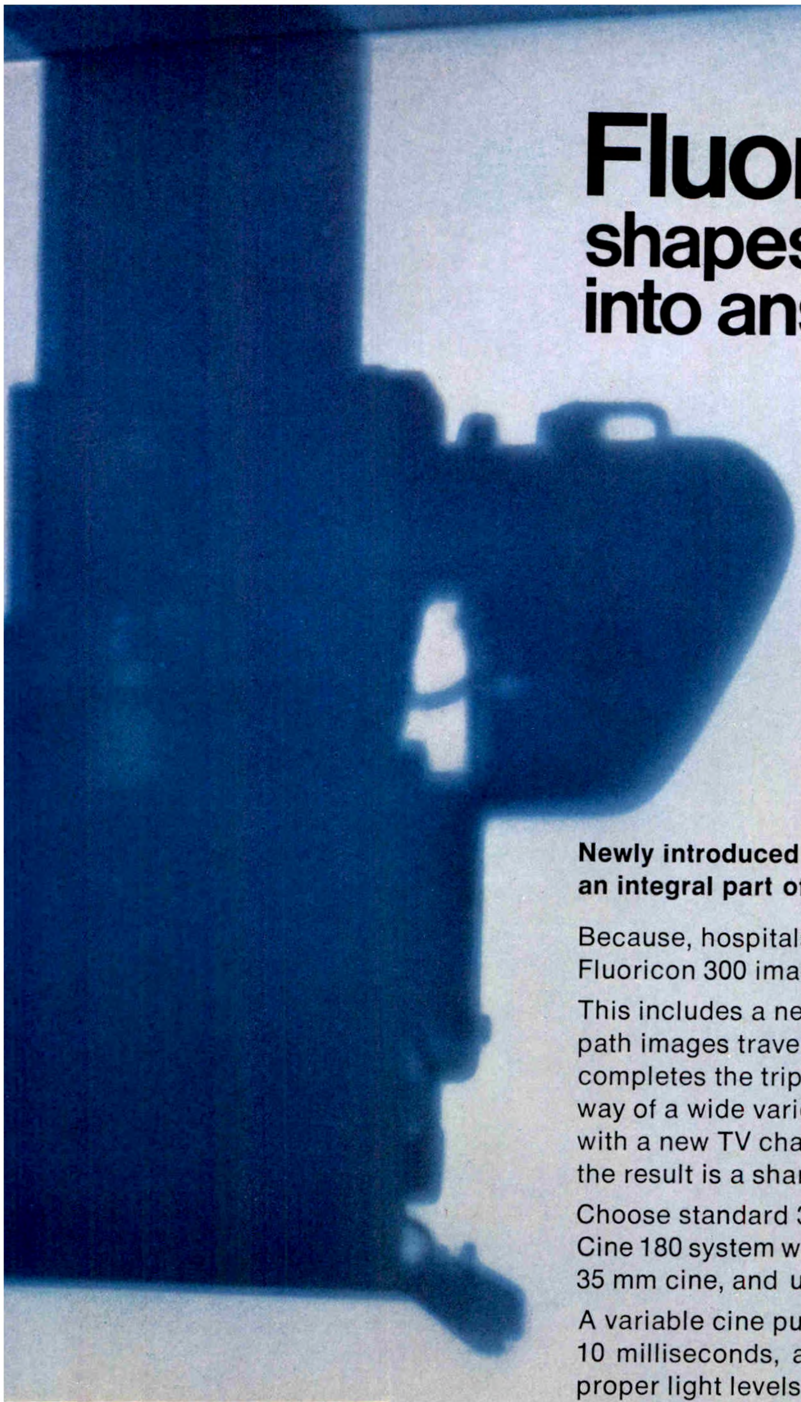

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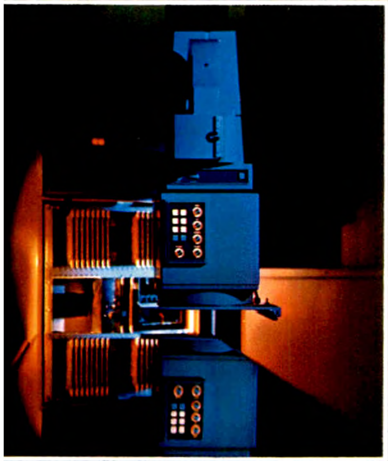

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2. Improved Patient Care and Flow
This efficient film handling system reduces interruptions to RT/patient contact. Lugging cassettes to the darkroom is greatly reduced. Loading and unloading film by hand become bygone chores. The time saved is spent in patient contact. Also, since film handling is now much faster and easier, examinations are completed more rapidly and patient flow is speeded.

3. Eliminate Darkroom
Some darkrooms can be eliminated since the Daylight System allows RTs to load packaged film in the dispenser, load film into cassettes, and process films—all in daylight conditions. The processor does not have to be in a darkroom; it can be elbow-close to the diagnostic room. This system, with various processing options, will handle one sheet at a time, or it can accumulate any number up to 50 exposed sheets and process them automatically. Again, all in daylight.

4. More Work in Same Space
This increase in film-handling speed will increase efficiency and capacity. How much depends upon your individual operation. Some people estimate up to a 30% improvement in efficiency. Therefore, you may not have to expand your department physically to handle an increased workload.

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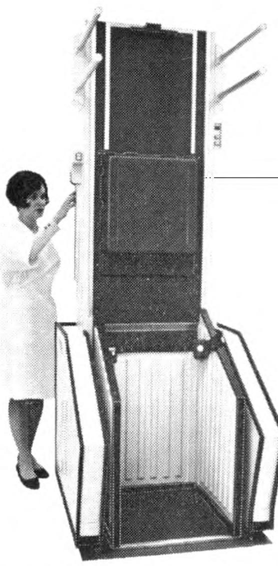

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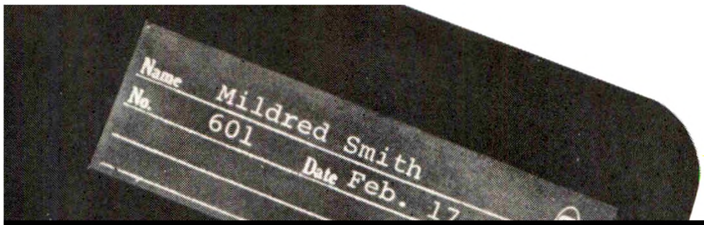

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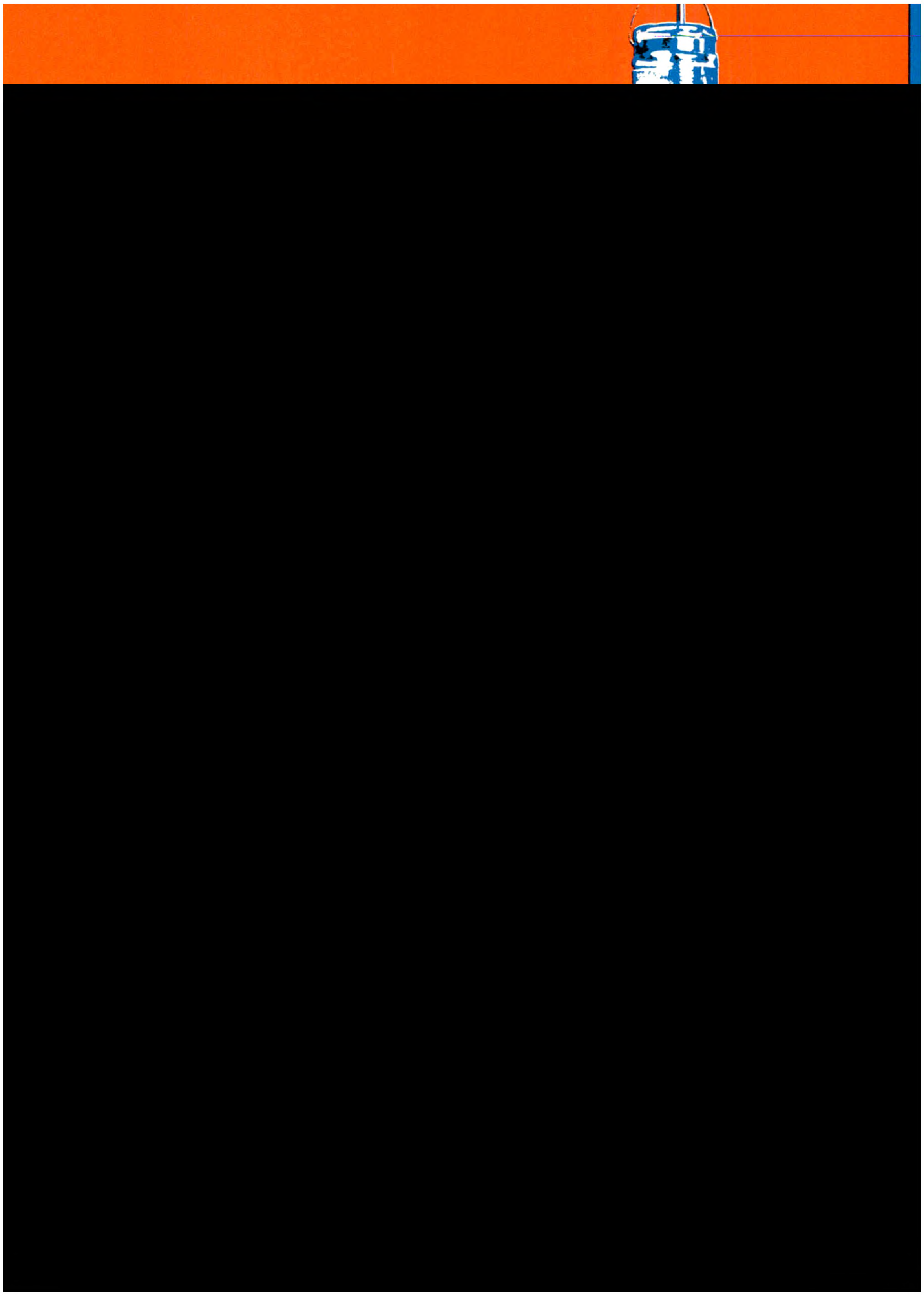

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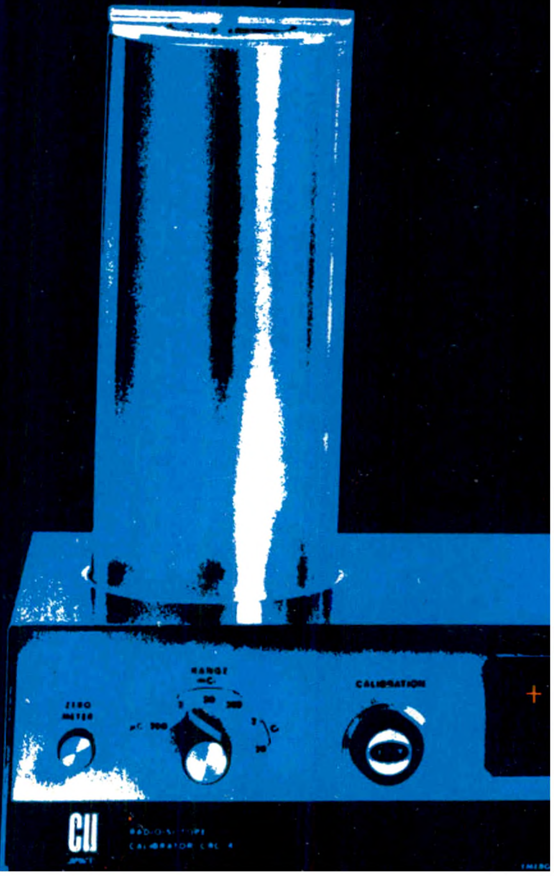

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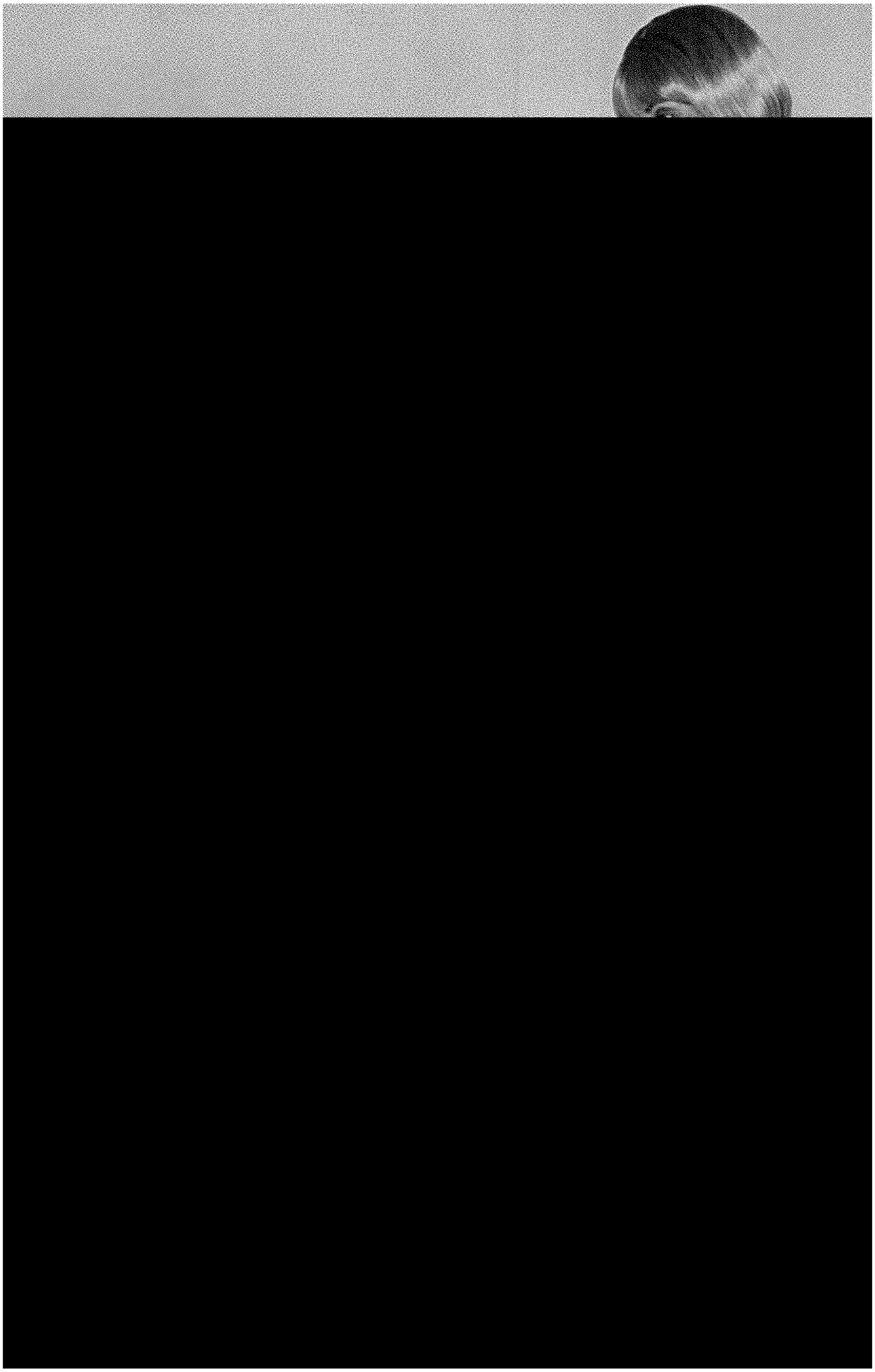

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TABLE I

	Os Centrale (one or more)	Extra Distal Carpals	Os Triangulare	Irregular Carpal Margins	Abnormally Shaped Scaphoid	Absent or Hypoplastic Scaphoid	Scaphoid Fused to Other Carpals	Abnormally Shaped Capitate	Absent or Hypoplastic Capitate	Some Carpal Fusion	Decreased Carpal Angle	Increased Carpal Angle	Maturation Disproportion	Diminution in Size of the Carpus
Arthrogryposis			x		x		x	x		X		X		X
Diastrophic dwarfism		X		X	x		x	x		x		x	X	X
Dyschondrosteosis										x	X			
Ellis-Van Creveld syndrome										X				
Epiphyseal dysplasia				X	x			X	X			X	X	X
Fanconi's anemia					X	X								
Hand-Foot-Uterus syndrome	X				X		X			X				
Holt-Oram syndrome	X		x		X	x	X	x		x				
Homocystinuria								X					X	
Oto-Palato-Digital syndrome	x	x			x		x	X		x				x
Symphalangism							x			X				
Trisomy 18													X	
Turner's syndrome										x	X			

X = Commonly present.
x = Occasionally present.

normalities of hands, feet and, in females, duplication of the genital tract. Clinically these individuals have very small feet with short great toes. The thumb is usually quite small. The roentgenologic findings in the foot include short first metatarsal with pointed distal phalanx of the great toe. The calcaneus is short and tarsal fusions are common. In the hand the first metacarpal is short and there is clinodactyly and brachymesophalangia of the 5th digit. An os centrale was present in 4 out of 10 hands of

Abnormality of the scaphoid is present in many of the congenital malformation syndromes. The reason this bone is so frequently involved is not clear. Minor abnormalities of the scaphoid are difficult to interpret since it is normally a variable bone.[27] A notch in its radial aspect may be normally seen[59] as can a bipartite scaphoid[2] which must be differentiated from pseudarthrosis.

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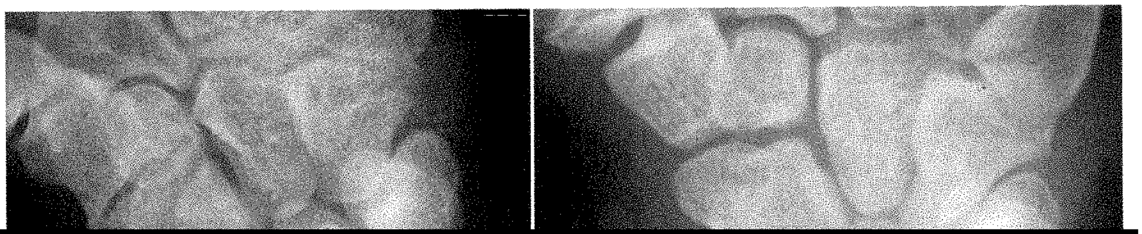

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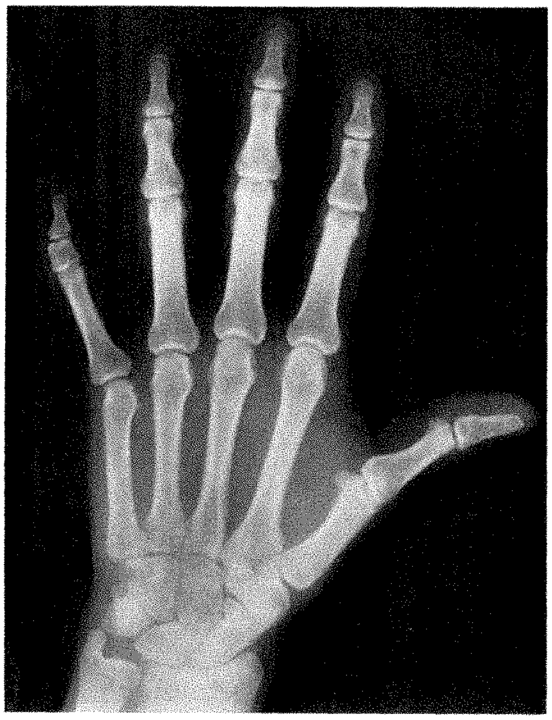

Fig. 4. Hand-Foot-Uterus syndrome. There is fusion of the trapezium and scaphoid. A well defined os centrale is seen. Note also the clinodactyly and brachymesophalangia of the 5th finger and the relatively short thumb.

tients the carpus appears smaller than usual.

FANCONI'S ANEMIA AND RADIAL HYPOPLASIA SYNDROMES

Hypoplasia of the scaphoid is commonly present in cases of radial hypoplasia (Fig. 6), except when associated with the Holt-Oram syndrome. The radial hypoplasia may include hypoplastic or absent thumb, thenar hypoplasia, hypoplasia of the distal radius, or absence of the radius. Radial hypoplasia is commonly associated with other anomalies besides aplastic anemia. Associated lesions include congenital heart disease, eye and ear abnormalities. Birch-Jensen[4] felt that prognosis in patients with radial defects is generally grave.

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defects include hypoplasia of the thumb with or without absence of the radius.[25] Clinodactyly of the 5th finger is frequently present.[47] Usually the scaphoid is hypoplastic or absent although Minagi and Steinbach[38] reported a case where the scaphoid was hypoplastic at 8 years but became unusually long at the age of 13 years. The usual fate of these scaphoid bones in later life is not clear since most patients die during childhood.

CONDITIONS ASSOCIATED WITH ABNORMALITY OF CARPAL ANGLE

The carpals in Turner's syndrome are often dysplastic. The normal gentle arch of the proximal bones becomes more V-shaped than usual (Fig. 7*A*) and in the most extreme form a Madelung-type deformity occurs (Fig. 7, *B* and *C*).[10,28,29] A quantitative evaluation of this arch was suggested by Kosowicz who described a decrease in the carpal angle in Turner's syndrome. The carpal angle was defined as the angle between two lines; the first tangent to the proximal scaphoid and lunate, the second one to the proximal triquetrum and lunate. The normal angle measures 131.5° (standard deviation 7.2°). In 55 per cent of patients with gonadal dysgenesis evaluated by Kosowicz, the carpal angle was reduced more than 2 standard deviations. A similar reduction occurred in 6 per cent of female controls. The carpal angle change is not evident in young children but becomes more manifest when the carpals become more mature. Kosowicz felt that these signs were already positive at the age of 6 or 10 years. Less frequent incidence of decreased carpal angle in Turner's syndrome was noted by Preger *et al.*[51] who found angles of less than 120° in only 6 out of 23 chromatin negative patients and in only 1 of 11 chromatin positive cases. Fusion of carpal bones has also been described. Finby and Archibald[10] had 2 examples in 33 cases.

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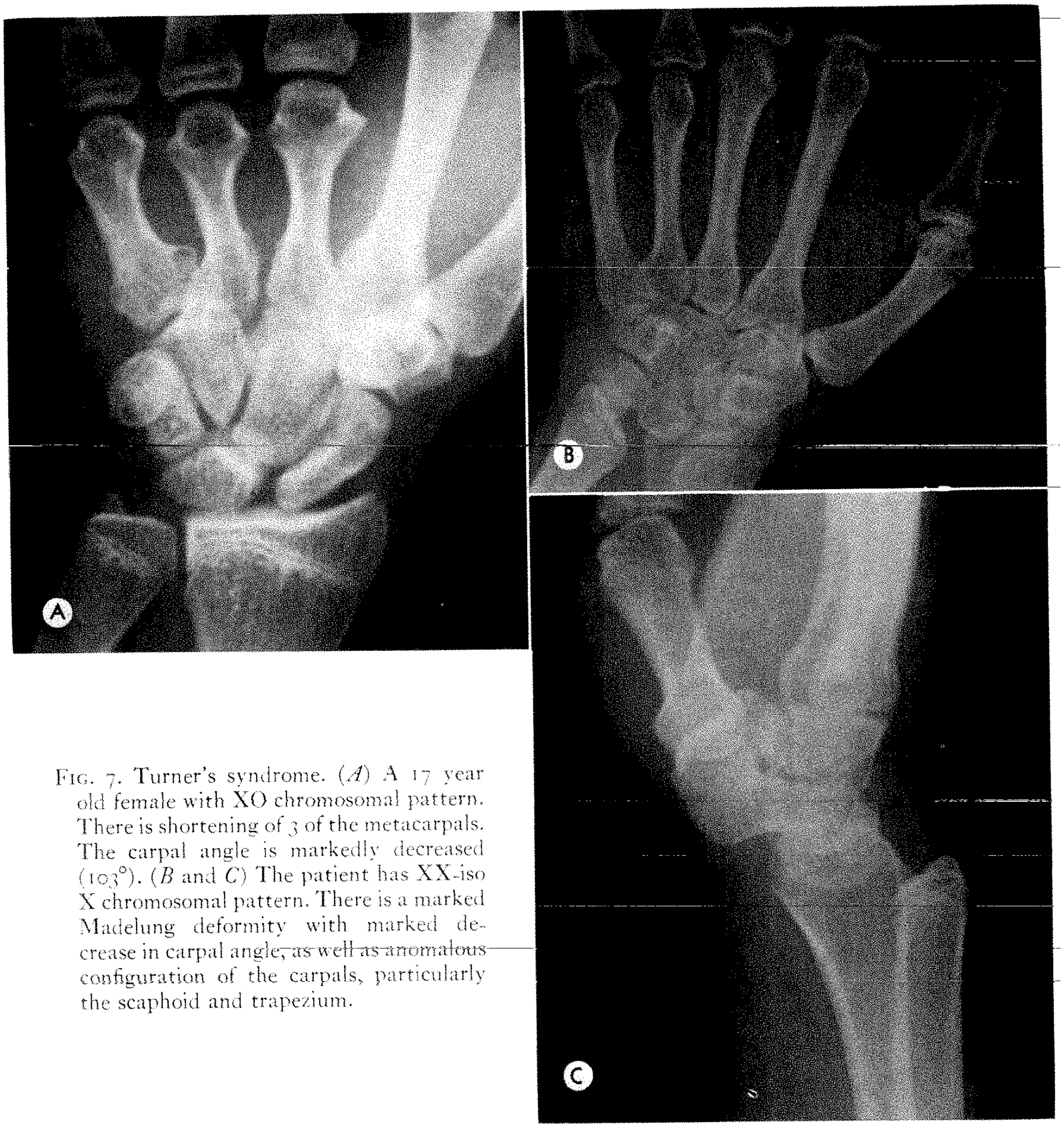

FIG. 7. Turner's syndrome. (*A*) A 17 year old female with XO chromosomal pattern. There is shortening of 3 of the metacarpals. The carpal angle is markedly decreased (103°). (*B* and *C*) The patient has XX-iso X chromosomal pattern. There is a marked Madelung deformity with marked decrease in carpal angle, as well as anomalous configuration of the carpals, particularly the scaphoid and trapezium.

in size to the other carpals or remain quite hypoplastic.[54] Silverman[54] described a patient who in earlier life had a roentgenologic picture of chondrodystrophia calcificans congenita, but later was typical of epiphyseal dysplasia. At the age of 16 years the distal row of carpals was hypoplastic with absence of the capitate and trapezoid. The carpals in the older patient also appeared diminished in size and the carpal angle was increased in many of our cases (Fig. 10*C*). This finding is also noted on reproductions of other cases reported in the literature.[24,54] The fragmentation of ossification in the carpal region has also been described in cretinism[27,65] but the conditions are clinically easy to distinguish. Irregular and delayed carpal ossification centers have been described in Morquio's disease, where delay in scaphoid ossification has been noted.[32] There is often a V-shaped configuration to the distal radius and ulna[32]

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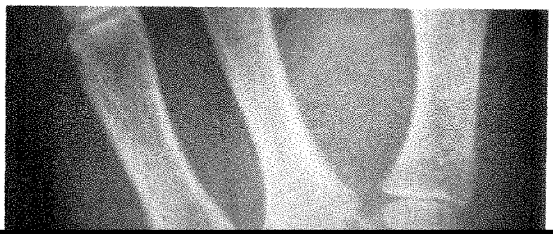

SYMPHALANGISM

Symphalangism is a hereditary condition which is characterized by partial or total absence of interphalangeal joints together with tarsal and carpal fusions. A variety of fusions have been described (Fig. 14). The

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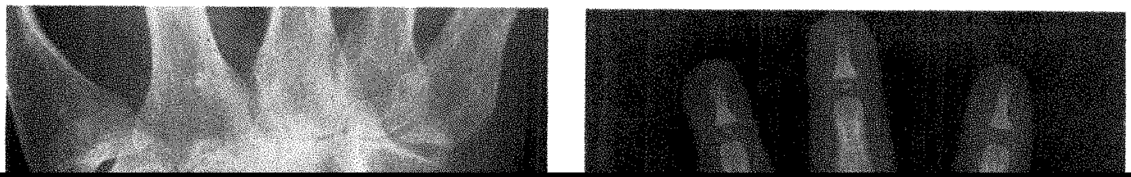

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TUBERCULOUS DACTYLITIS IN THE ADULT*

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THE short tubular bones of the hands and feet are uncommon loci for the manifestations of adult skeletal tuberculosis. Tuberculous dactylitis, in particular, is considered a hallmark of childhood and our roentgen concept of this lesion, as described in texts and major works on tuberculosis, has been largely that of spina ventosa. Scattered reports additionally imply its rarity in the adult.

We, therefore, believe that our experience with 23 adult, biopsy proven cases of tuberculosis distal to the tarsus and wrist merits presentation. This report's prime purpose, however, is not to serve as a review of skeletal tuberculosis, but rather to call attention to its occurrence in infrequently documented areas, to emphasize its morphologic variability in these locations, and to illustrate a variety of little emphasized roentgen manifestations.

STATISTICS

Reports of skeletal tuberculosis have chiefly come from general surgical, orthopedic and pathologic files. Among the larger series stemming from sanitaria, LaFond[26] noted a 5.2 per cent gross skeletal incidence among 6,682 adult admissions for active disease (6 cases involved the hand, 9 the foot), while Auerbach and Stemmerman[3] found a 10.4 per cent incidence (119 of 1,143 autopsies) including 5 metatarsals, 4 metacarpals and 1 phalanx with ages unstated.

Tuberculous dactylitis statistics *per se*, for the most part, embrace the infancy to childhood period, ranging from 0.6 to 14 per cent in childhood, and varying with clinical[15] or surgical[18,38] cases. In the larger childhood series,[18,38] dactylitis became uncommon after 5 and scarce after 10 years. A recent surgical report of hand and wrist tuberculosis [34] included 17 dactylitis cases; 9 were in children and 8 in adults at the time of onset. A Mayo Clinic musculoskeletal tuberculosis review [21] included 27 bone and joint cases; 3 occurred solely in hand tendon sheaths and none in the hand or foot skeleton. Not one child was seen in the 5 year period. Although other isolated reports of adult peripheral skeletal tuberculosis as herein defined (*i.e.*, distal to tarsus and wrist) were found, [1,2,10,16,26,34] radiologic reviews have been rare.[29,31,36,40,41]

Poppel *et al.*[31] observed 7 cases of small tubular bone tuberculosis among 156 with skeletal involvement (the 3 illustrated were adults), Stenstrom[40] 3 phalangeal cases in adults 29, 69, and 76 years old, while Nathanson and Cohen[29] in a roentgen review of 200 Seaview Hospital cases, found 17 metacarpals, 8 metatarsals and 14 phalanges involved among 100 children (1–16 years) and 1 metacarpal, 1 metatarsal and 0 phalanges among 100 adults (16–70 years).

SEX AND AGE

Most adult series note a male preponderance (3:1 ratio)[3,26,29,33] with no prominent sex difference when skeletal loci or extent of lesions are considered. Although most adult series give a second and third decade peak incidence of bone and joint tuberculosis, apparent discrepancies result from comparing clinical with pathologic and autopsy findings. In several large clinical series,[45] the age incidence of skeletal tuberculosis ranged from 69 to 85.3 per cent in the first two decades of life, while autopsy series ranged from 49.6 per cent (20–39 years)[2] to 79 per cent over age 20.[4,8] Contrasting incidences may, in part, be attributed to the

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fact that the pathologist usually sees progressive cases, and at a much later stage and age than the clinician.

Among Nathanson and Cohen's[29] 100 adults, skeletal tuberculosis was most frequent at those ages at which physical activity is most strenuous; *i.e.*, 77 per cent between 16–45 years, 23 per cent after 45 years, with a peak between 20–25 years, a gradual decline till 40–45 years and an abrupt drop thereafter. Among the small tubular bone cases of Poppel *et al.*,[31] the 3 detailed were in adult males 28, 36 and 62 years of age.

MATERIAL

Among 200 adult skeletal tuberculosis cases in our surgical and orthopedic pathology files (1930 to 1970) the ages ranged from 17 to 90 years; 10 occurred in the hand distal to the wrist, and 13 in the foot distal to the tarsus. Of the 10 hand cases, 4 had soft tissues alone involved (*i.e.*, tendon sheaths), 3 the metacarpals and 3 the phalanges. Of the 13 foot cases, 3 had soft tissues solely involved, 6 the metatarsals, and 4 the metatarsal-phalangeal joints.

There were 11 males and 12 females (Table 1).

Table I

SITE AND SEX DISTRIBUTION OF 23 CASES OF PERIPHERAL SKELETAL TUBERCULOSIS
(Distal to Carpus and Tarsus)

	Total	Male	Female
Hand			
Soft tissues	4	1	3
Metacarpals	3	2	1
Phalanges	3	1	2
Foot			
Soft tissues	3	2	1
Metatarsals	6	2	4
Metatarsal-phalangeal joints	4	3	1
	23	11	12

PATHOPHYSIOLOGY

Although it is generally held that skeletal involvement occurs chiefly via the hematogenous route, the origin and onset of the bacillemia and the mode of the organism's skeletal localization have been controversial subjects and several salient concepts deserve mention.

MODE OF LOCALIZATION

König[26] held that osseous involvement was due to obstructive emboli to nutrient vessels resulting in tuberculous infarcts. His tenets have been used to explain the metaphyseal predilection of tuberculosis in children's long bones; *i.e.*, since "the metaphyseal arteries' terminal branches are very small, bacilli tend to lodge here." Others,[2,17] unable to find tuberculous plugs occluding afferent arteries in man, attributed the impaired blood supply of involved areas to an obliterative endarteritis. They ascribed the occlusion to an intimal thickening of loose *non-tuberculous connective tissue* resulting from toxic irritation by the tuberculous process rather than to a tuberculous plug.

Origin and onset of the bacillemia have been other points of contention. Auerbach[2,3] relates most skeletal involvement to hematogenous dissemination during the florid phase of the primary complex with the fate of "seedings" being determined by the primary's evolution. With healing and/or encapsulation of the primary complex, there is a tendency toward resolution of skeletal foci, most healing without demonstrable residua. An occasional focus, however, may persist, pursue an indolent course, and become clinically or roentgenographically evident years later. The reason for localization of active disease to one area when multiple areas had been infected remains unclear. Some authorities[31] have invoked the "loci minoris resistentiae" theory; *i.e.*, infected foci remain indefinitely dormant, then, following decreased local resistance, such as may occur with trauma or generalized debility, they are capable of reactivation. Others contend that adult skeletal disease is due to sporadic hematogenous dissemination at a later date, either from a quiescent primary or from an extra-osseous focus.

Skeletal lesions in most children, however, originate soon after the primary infection.

In any event, bacteremia and widespread implantation must have occurred in any case having even a solitary bone or joint lesion, and small bone involvement, like osseous tuberculosis elsewhere, is due to hematogenous infection, whether occurring in childhood or adulthood, with widespread visceral and/or osseous disease, or as a solitary skeletal lesion unaccompanied by other stigmata of tuberculosis.

ASSOCIATED PATHOLOGY

Associated tuberculous foci are sought as aids in corroborating skeletal diagnoses or as origins for bone lesions. The incidence of associated visceral disease, however, (*i.e.*, active or chronic pulmonary and/or renal disease) varies with age range and material analyzed (*i.e.*, clinical, roentgenologic, surgical or autopsy).

It has been held that osseous tuberculosis and active pulmonary tuberculosis do not commonly co-exist; however, the frequency of roentgenographically diagnosable associated disease, particularly of the lungs, is not easily arrived at. In Reisner's[33] clinical study, 53 per cent of 160 skeletal tuberculosis cases had "negative lungs." Discrepancies between lower clinical and higher autopsy "positive" pulmonary statistics may be due to the fact that frequently terminal miliary cases were omitted from clinical series. In addition, apical foci were often roentgenographically unrecognized, particularly in the presence of perifocal emphysema. Interestingly, Reisner[33] found 20 per cent with chronic pulmonary tuberculosis, while Auerbach[2] noted 19.3 per cent at autopsy. Auerbach also found concomitant urogenital tuberculosis in 49 of 119 cases with skeletal disease. Among 132 other autopsies with Pott's disease,[3] an active chronic pulmonary process was found in 35 (26 per cent), with urogenital lesions coexisting in 38 (29 per cent). Although deemed possible that hematogenous dissemination from the lungs or urogenital system might have occurred, *clinical and pathologic data most often indicated that the skeletal tuberculosis was the older process.* In 80 cases (66 per cent), no fresh source of hematogenous dissemination to the spine was found; however, old pulmonary, hepatic, and splenic foci re-enforced Auerbach and Stemmerman's[3] belief that skeletal involvement is most frequently related to the primary complex.

Among the Mayo Clinic's[21] 27 adults with musculoskeletal tuberculosis, 3 had clinical evidence of acute and 10 of past pulmonary tuberculosis. Poppel *et al.*,[31] among 156 skeletal cases, found 20 per cent with active pulmonary lesions, and 20 per cent with coexistent genitourinary tuberculosis; 8 cases had combined skeletal, renal and pulmonary tuberculosis. It is not specified, however, how many of these lesions were detected roentgenographically.

Mann[27] noted history, symptoms and bacteriologic studies to be of little assistance in discovering early lung lesions among 500 skeletal cases and relied on roentgenograms, 284 (57 per cent) of which showed active pulmonary infiltrates. However, 84 per cent of positive studies were in younger patients. He noted that the younger the child, the more likely are primary pulmonary infiltrates, enlarged draining mediastinal lymph nodes and bone lesions to be found simultaneously. In the older child and adult, by the time the skeletal lesion is manifest, the pulmonary infiltrate has healed. Therefore a negative chest roentgenogram in the adult is of little value, while in the younger age groups it casts doubt on the tuberculous nature of the skeletal lesion.

Among the 3 detailed adult dactylitis cases of Poppel *et al.*,[31] 2 had associated lesions; a 36 year old male had metacarpal and sphenoidal wing tuberculosis, while a 28 year old male with a metacarpal-phalangeal lesion later manifested lumbosacral and pulmonary tuberculosis. A third, 62 year old male, had sole metacarpal disease. Among Robins'[34] 17 tuberculous dactylitis cases, 10 had associated lesions, only 1 being pulmonary. Nathanson and

Cohen[29] also commented upon "infrequent association of osseous and active pulmonary tuberculosis," but noted the reverse to be true in their series with 55 per cent pulmonary infiltration. They did not indicate, however, the number diagnosed roentgenographically.

Among our 23 cases, 2 of 17 patients with roentgenograms available had evidence of active pulmonary disease and 1 had active renal and bladder tuberculosis.

MULTIPLICITY OF SKELETAL LESIONS

Among the 100 adults of Nathanson and Cohen,[29] 72 had single and 28 multiple bone involvement. Poppel *et al.*[31] had 12 cases with multiple osseous foci (including 1 with dactylitis and sphenoid ridge involvement); 10 were asymptomatic. Among Auerbach's[2] 119 cases, 1 part of the skeletal system was affected in 86 and 2 or more in another 33.

It has been stated[41] that tuberculous dactylitis *per se* is more frequently multiple in childhood, being more often associated with generalized tuberculosis. Among our 23 adults, 1 case had a metatarsal and 1 a metacarpal involved (Table II). Of the remaining 21 cases, 6 had multiple osseous foci, none of which were peripheral, 9 had dactylitis as the sole lesion, while in the remaining 6, this information was not available.

REPORT OF CASES

Case I. G.V., a 42 year old Puerto Rican female, was seen on November 6, 1969 with the chief complaint of swelling of left thumb of 5 weeks' duration with no antecedent trauma. She had been treated elsewhere with penicillin injections with no improvement. She had been in prior good health. Two siblings died of tuberculosis in childhood.

Physical examination revealed a red, tender, fluctuant mass over the metacarpal-phalangeal joint of the thumb. Aspiration yielded 10 cc. of pus.

Roentgenograms (Fig. 1*A*) were interpreted as showing osteomyelitis. Intramuscular and oral penicillin were prescribed. Subsequent roentgenograms (Fig. 1, *B* and *C*) showed no response to treatment. Cultures from aspirations revealed *Staphylococci* (coagulase positive and negative).

Surgery on December 4, 1969, disclosed bony spicules in the soft tissues and a softened proximal phalanx.

Microscopic examination showed a granulomatous osteitis of the tuberculous type. Stains for fungi and acid fast bacilli (AFB) were negative. Routine cultures grew out *Staphylococci* (coagulase positive), sensitive to oxicillin.

The laboratory findings are given in Table II. An intermediate PPD was positive. Coccidioidin and histoplasmin skin tests were negative.

Intravenous pyelography, chest roentgenography, and skeletal survey were normal.

Postoperatively, she had a fever of 102° F. which subsequently decreased to 100° F. where it remained for the duration of her hospital stay. She was treated with oxicillin 600 mg. every 6 hours.

On December 9, 1969 a second fluctuant abscess developed over the radial side of the base of the left thumb which communicated with the previous ulnar-sided incision. Incision yielded 20 cc. of pus. The abscess again extended to the bone. A gauntlet cast was applied.

Roentgenograms (Fig. 1*D*) showed further marked bone destruction with pathologic fractures and sequestrations within the proximal phalanx as well as deossification of the head of the first metacarpal and distal phalanx. Routine cultures showed no growth. Two sputum cultures, 3 urine cultures, 4 smears, and 2 cultures from the abscess were negative for AFB, but a third abscess culture and a biopsy specimen were positive.

She was placed on triple therapy; the wound drainage gradually decreased and she was discharged on January 3, 1970 to the Plastic Surgery Clinic. After removal of the cast on May 4, 1970 there was suggestive evidence of a pseudarthrosis and the drainage had ceased by May 11, 1970.

She left the country on July 3, 1970, while still on antituberculous drug therapy, with a minimal residual deformity of the thumb (Fig. 1*F*).

Case II. J.S., a 21 year old Puerto Rican housewife, was admitted on March 6, 1956 with the chief complaint of swelling of the dorsum of the right foot for 11 months. There was no his-

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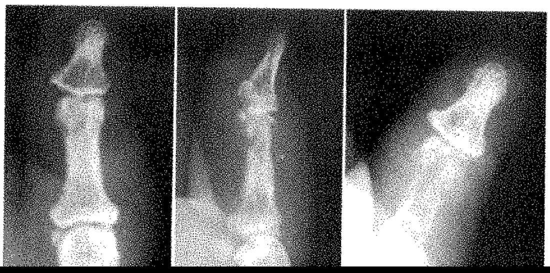

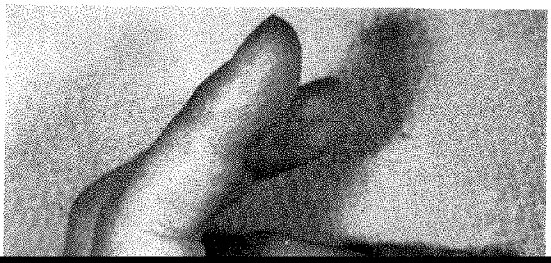

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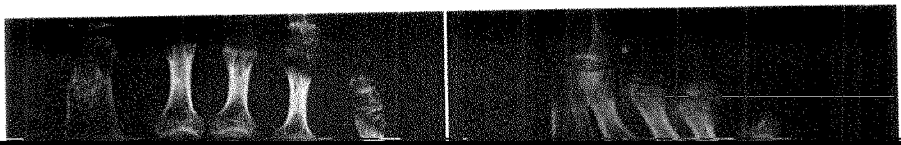

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A second operation on February 23, 1961 drained another fluctuant abscess below the

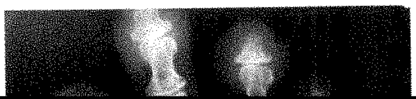

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Table III

ADULT *vs.* CHILDHOOD TUBERCULOUS DACTYLITIS

	Child	Adult

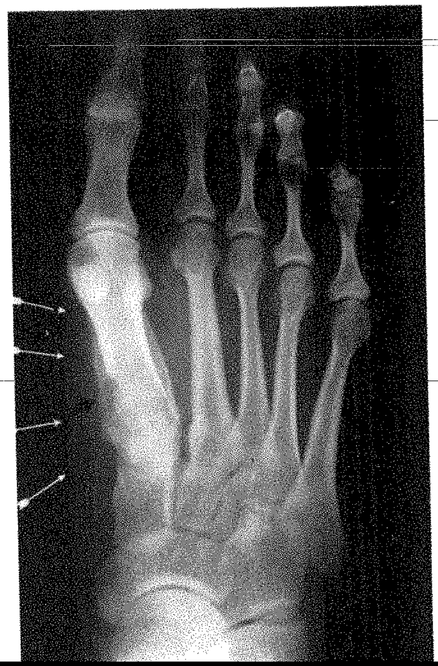
children, when the marrow becomes extensively infiltrated with proliferating tuberculous granulation tissue, the epiphysis remaining uninvolved (Fig. 7, *A* and *B*; and

[redacted]

superimposed pyogenic infection or an infected sinus tract.

Although these statements may be true in large numbers of cases, departures are so frequent that they must be considered more than exceptions to the rule. Contrary to the dictum that osseous tuberculosis is purely or diffusely destructive, well circumscribed or punched-out appearing cystic lesions may be encountered which vary widely in appearance. They may occur as solitary or multiple circumscribed, radiolucent defects (Fig. 5) confined to a localized area within, or widespread throughout the shaft of a single tubular bone (Fig. 6, *A* and *B*). Considerable bone production may develop about these localized rarefied areas.

Along with local reactive sclerosis, either fine periosteal proliferation or profuse



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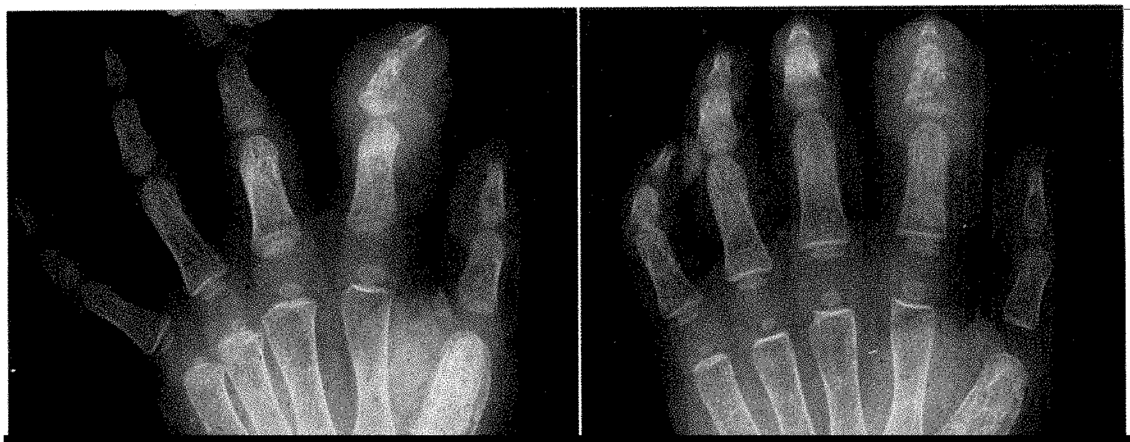

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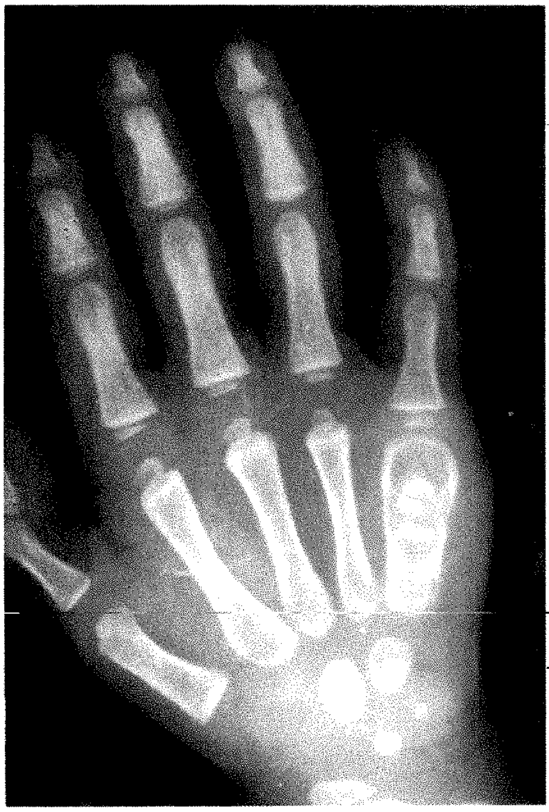

FIG. 9. Child. Spina ventosa. Note central destruction and stippling, cystic expansion, and epiphyseal sparing.

noted. Fistula formation commonly seen in the child occurred in 6 of our cases.

The roentgen changes of peripheral osseous tuberculosis, as herein defined, may, therefore, be grouped into 4 broad categories:[7,14]

1. *Soft tissue swelling.* This often is marked and fusiform and may be the sole manifestation with no apparent change in neighboring bones or joints and, in children, may resolve completely.

2. *Periostitis, expansion, cortical and cancellous destruction—spina ventosa.* The earliest roentgen evidence of bone involvement in child and adult (Fig. 1, *A–E*), is a periostitis indicated by a linear deposit of new bone predominantly involving the diaphysis in the child (Fig. 8*B*) or any portion of the adult small tubular bone. The periostitis may intensify with increasing cortical thickness and expansion. It may be so well defined that the original shaft is distinctly evident within the periosteal sheath of new bone (Fig. 2, *A* and *B*; 7; and 8*B*). Gradual destruction with sequestration of the former then occurs while the involucrum thickens (Fig. 5). If much of the involucrum's internal aspects are absorbed, a cryst-like cavity remains which appears to be ballooned out or injected with air, the so-called spina ventosa (Fig. 9; 10; and 11). Concomitantly, dense stippling of one or more areas may be seen so that the involved bone, in addition to appearing twice its normal diameter, may be of greater density, stippled and without normal cancellous trabeculae (Fig. 10; 11; and 13*A*). The above changes, particularly in children, may resolve, the bone being so completely restored as to be indistinguish-

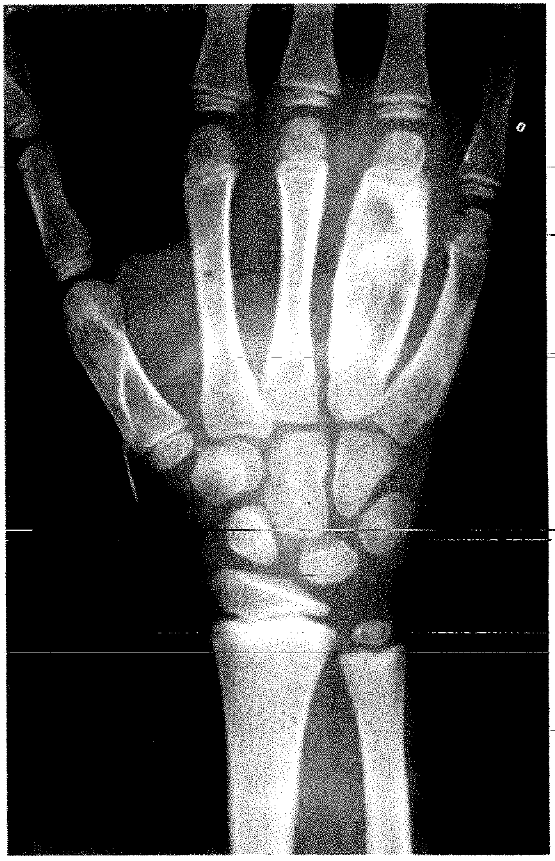

FIG. 10. Child. Spina ventosa. Note ballooning of 4th metacarpal, reactive osteitis, mixture of internal sclerosis and destruction. Epiphyseal sparing.

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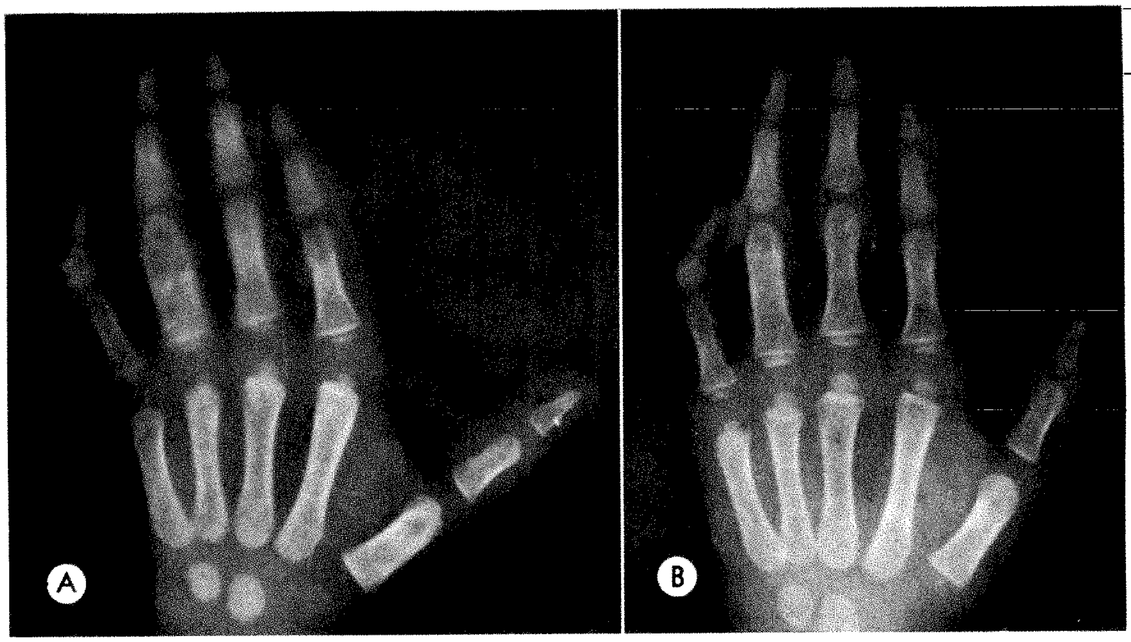

Fig. 12. (*A*) Child. Involvement of the 1st metacarpal, proximal phalanx of the fourth finger and middle phalanx of the fifth finger. (*B*) Four and one-half months later. Almost complete resolution and normal appearance at all sites. (Courtesy of Dr. I. Katz, Brooklyn, N.Y.)

to impaired blood supply due to obliterative endarteritis.[11] The involved dead bone roentgenographically appears either of normal or increased density due to lack of resorption in the former case or subsequent reactive osteitis and recalcification in the latter.[30] Auerbach and Stemmerman[3] demonstrated productive changes in vertebral autopsy specimens which, during life, showed extensive increased density roentgenographically and noted 2 pathologic types of osseous tuberculosis to explain observed roentgen variations; *i.e.*, the productive or sclerotic form and the destructive, caseous or exudative form.

Grossly, in the *productive form*, the bone is firm throughout, maintaining morphologic integrity despite total vertebral body involvement. Microscopically, there is no actual destruction but rather trabecular absorption;[3] *i.e.*, tuberculous granulation tissue, which fills the marrow spaces, erodes neighboring trabeculae leading to bone atrophy. Residual trabeculae are accentuated resulting in the over-all coarsened (Type 3) trabecular pattern. Small areas of caseation, walled off from the trabeculae by productive elements, may or may not be present. Bone density, therefore, varies with the amount of granulation tissue and caseous material within the marrow interstices as well as with the amount of reactive osteitis present. Axial lesions of this type are extremely difficult to delineate roentgenographically; *i.e.*, the intermingling of destructive and reactive processes may balance each other so effectively as to prevent roentgen detection of extensive bone damage. These subtleties are more readily appreciated in the peripheral skeleton, however.

In the *exudative form*, large areas of caseation have completely destroyed normal bony architecture. Sclerosis due to reactive osteitis or periostitis as well as calcification is minimal or absent. Marked softening of bone, trabecular resorption and caseation leading to suppuration and liquefaction, through digestion by polymorphonuclear proteolytic enzymes, develop. Abscesses so formed dissect towards the skin forming fistulae which extend for vari-

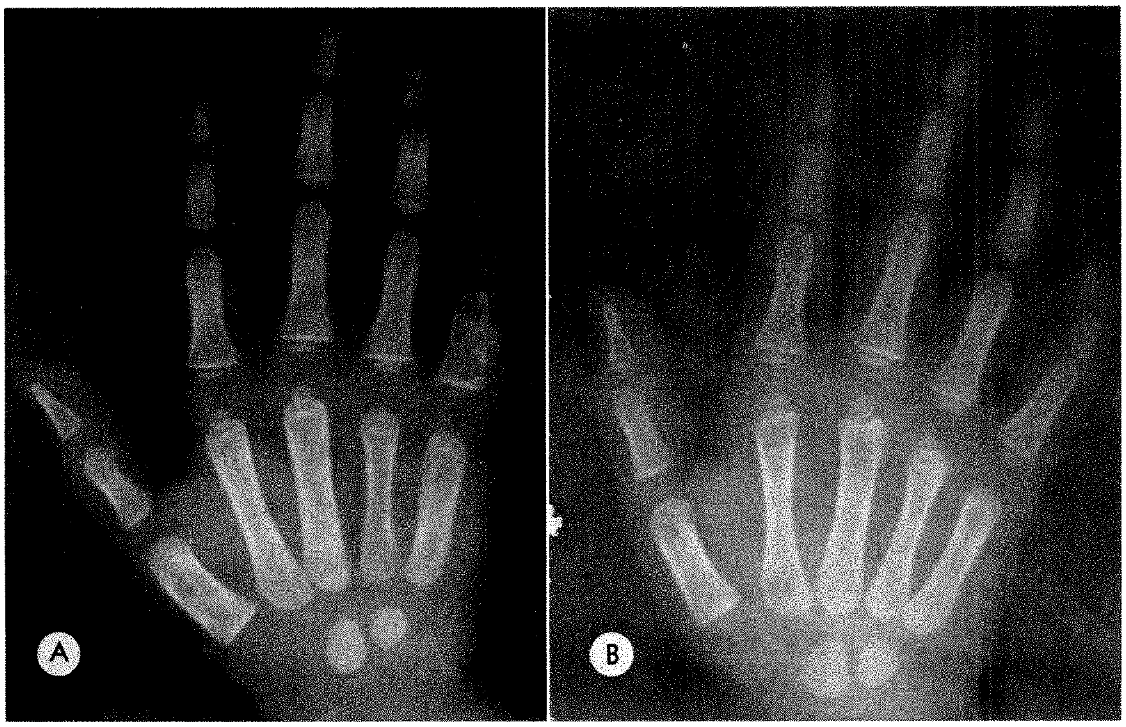

Fig. 13. (*A*) Child. Minimal involvement of 1st metacarpal with more obvious manifestation at 4th metacarpal and proximal phalanx. (*B*) Four and one-half months later. Complete roentgen resolution.

able distances with sinus ramification always greater than can be clinically appreciated from the size of a sinus opening or amount of discharge.

Abscess formation, in addition to bony fragmentation, may result in periosteal stripping and periostitis at a slowly progressive pace. However, with sudden stripping of periosteal elements, devascularization and bone death may occur without obvious roentgen change; *i.e.*, a still grossly intact bone if rapidly deprived of blood supply remains inert without change in specific gravity upon which the roentgen ap-

to local denervation by perineural fibrosis to neural compression by caseous masses and to actual destruction of nerves passing through disease areas. Periosteal stripping also causes loss of innervation as well as blood supply.

DIFFERENTIAL DIAGNOSIS

Since adult tuberculous dactylitis is rarely reported, roentgen criteria are difficult to establish and several simulators require differentiation. Syphilitic dactylitis is probably the most difficult to exclude. In infants and children it tends to be bilat-

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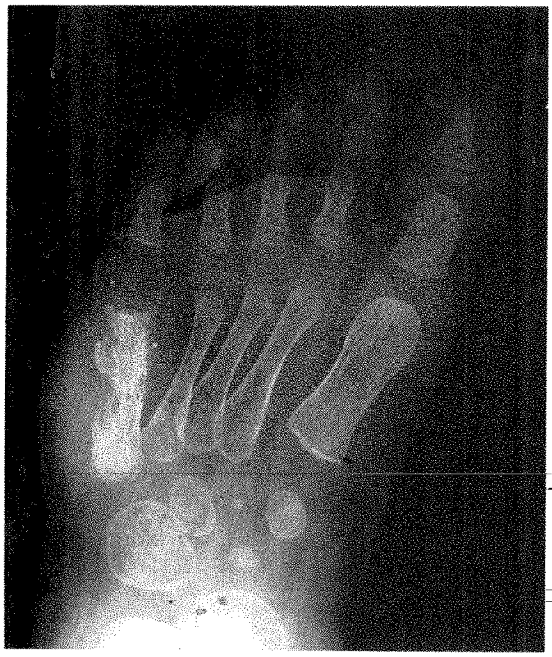

Fig. 14. Child. Profuse periosteal reaction with lateral cortex interrupted, sequestration and soft tissue swelling. Note reactive osteitis rather than demineralization.

of syphilis elsewhere, particularly in the long bones, a rare occurrence in tuberculosis, is helpful.

Small tubular bones are a favorite site for

carcinomatosis usually produce characteristic roentgenographic appearances simultaneously elsewhere. The manifestation of acute pyogenic osteomyelitis may be associated with obvious infection or traumatic causation and with more rapid sequestration, arthritis and clinical signs of sepsis. Pyogenic articular lesions secondary to osseous foci are much less common than isolated pyogenic osseous foci, perhaps due to the growth plate serving as a barrier to extension. Conversely, with tuberculosis, concomitant joint involvement is much more common than a purely osseous focus.[39] Therefore, tuberculosis is reputedly more commonly accompanied by joint involvement than is pyogenic osteomyelitis. Although bony and fibrous ankylosis may occur in both pyogenic and tuberculous arthritis, both are more common in the former, while fibrous ankylosis is more common in the latter unless secondary pyogenic infection supervenes.

Mycetoma (maduromycosis), leprosy and yaws in nonendemic areas may be mistaken for tuberculous dactylitis, but are usually polyostotic with numerous soft tissue stigmata. Madura foot is preceded by local injury, whereby the fungi are introduced into

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dioidomycosis, actinomycosis and torulosis, to affect bony eminences such as the olecranon, malleolus and patella.[35]

present) most commonly yielded staphylococci or streptococci rather than acid fast bacilli

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chance of arresting the disease and preserving useful function, particularly in non-weight bearing areas.

SUMMARY

Tuberculous dactylitis, although most frequently encountered in children, also occurs in adults in whom its roentgen manifestations have not been sufficiently emphasized. The evolution of the disease is often subtle with far advanced changes present in some cases when first seen.

Tuberculosis of bones and joints, although decreasing in incidence, is still the most common granulomatous bacterial arthritis, with a peak incidence occurring in an older age group than is ordinarily assumed and with lesions occurring in "unusual" locations.

An analysis of our experience with 23 cases of adult tuberculosis distal to the tarsus and wrist is presented with a brief review of the literature and an emphasis on heretofore infrequently stressed roentgen changes.

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ARTHROGRAPHY OF THE RHEUMATOID WRIST JOINT*

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RHEUMATOID arthritis is a systemic disease primarily affecting the synovium and only secondarily producing the destructive changes at the subchondral bone seen on roentgenograms.

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duced dorsally into the wrist between the navicular bone and radius (Fig. 2); 1.5 cc. of meglumine diatrizoate (renografin 60) is injected under fluoroscopic control; and roentgenograms in anteroposterior, lateral

CLINICAL STUDY

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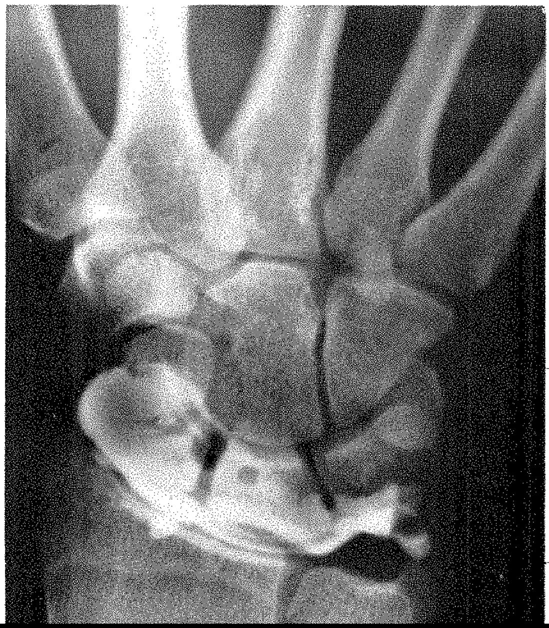

noted in communication between the radiocarpal and the midcarpal joints. This is caused by destruction and perforation of the triangular cartilage of the radiocarpal joints and/or the interosseous ligaments. Several arthrograms which showed filling of the midcarpal joints also showed filling of the carpometacarpal and intermetacarpal joint spaces of the second, third and fourth metacarpals. However, the carpometacarpal joint of the thumb was never opacified. In 21 per cent of the arthrograms there was a communication between the radiocarpal joint and the extensor tendon sheath. The extensor carpi ulnaris was outlined in 11 cases and the extensor digitorum communis in 2 cases. In one wrist the volar flexor tendon sheath was opacified (Fig. 9). Such communication results from the invasive

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raphy has a place in the routine diagnosis of rheumatoid arthritis. However, in early cases, it may demonstrate the abnormalities before changes are detectable on plain roentgenograms. In more advanced cases, arthrographic demonstration of the extent of synovial involvement has proved useful when synovectomy is planned.

SUMMARY AND CONCLUSION

The arthrographic findings in rheumatoid wrist joints are correlated with those in non-rheumatoid patients. Soft tissue abnormalities precede the subchondral bone changes seen on conventional roentgenography.

A combination of two or more of the listed arthrographic abnormalities tends to confirm the diagnosis of rheumatoid arthritis. The arthrographic information has been useful in planning the extent of synovectomy. Surgical exploration in 15 cases confirmed the abnormalities demonstrated on the arthrogram.

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THE AVULSIVE CORTICAL IRREGULARITY*

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UNUSUAL cortical defects of the distal femur in the area of the medial condyle were first reported in 1951 by Kimmelstiel and Rapp.[11] Since that time, 2 articles have appeared in the literature describing the roentgenographic appearance of these lesions.[2,14] The pathogenesis and true significance of these lesions, however, have not yet been satisfactorily explained.

No generally accepted or consistent name has been applied to the lesion which has been variously referred to as periosteal desmoid,[11] subperiosteal desmoid,[15] cortical desmoid,[10] subperiosteal abrasion,[4] cortical abrasion,[4] medial distal metaphyseal femoral irregularity,[14] and subperiosteal cortical defect.[1] Avulsive cortical irregularity is believed to be a more appropriate term for this lesion since the pathogenesis, general location and roentgenographic appearance are implicit in the name.

ROENTGENOGRAPHIC APPEARANCE

The avulsive cortical irregularity has a highly characteristic location and roentgenographic pattern. The lesion is almost exclusively observed on the posterior aspect of the medial femoral condyle along the medial ridge of the linea aspera just above the adductor tubercle[2,10,11,15] (Fig. 1, *A* and *B*). The described lesion is best demonstrated in the slightly oblique position with the lateral condyle projected anteriorly and inferiorly. In this projection, the lesion appears as a shallow, concave, irregular osteolytic area originating on the surface of the cortex (Fig. 1, *C* and *D*; and 2, *A* and *B*), and almost without exception, the long axis of the lesion parallels the long axis of the femoral shaft[1] (Fig. 3). Tiny cortical fragments are occasionally seen in the soft tissues adjacent to the lesion (Fig. 5*B*). The avulsive cortical irregularity may be as large as 4–5 cm. in length. In the true lateral projection, the lesion usually appears as a localized area of cortical roughening projected over the popliteal area[2] (Fig. 4). The avulsive cortical irregularity occasionally simulates a malignant lesion. Early in the process of development it may appear in the anteroposterior projection as a localized area of bony spiculation[14,15] (Fig. 5*A*). This is in sharp contrast to the well defined cortex normally observed in this area. In some cases the edge of the bone surrounding the defect is of normal density, but the lesion may have a sclerotic margin. The presence of sclerosis indicates the healing phase in the evolution of the lesion.[4,15] It is reasonable to assume that the completely healed lesion may appear as a localized area of cortical hyperostosis. These lesions are almost invariably more proximally located, since continued growth at the epiphyseal plate advances the lesion from the epiphysis (Fig. 6, *A* and *B*).

The avulsive cortical irregularity has been observed between the ages of 3 and 17, but is most commonly seen from 10 to 15 years of age. In the age group of 3 to 17 years, the lesion has been reported to occur in 11.5 per cent of males and 3.6 per cent of females[14] with approximately 35 per cent of the lesions present bilaterally.[9] After closure of the epiphysis, however, the unhealed lesion is extremely uncommon.[14] Most of these lesions are discovered incidentally when roentgenographic examination of the knee is performed because of trauma or other reason believed unrelated to the avulsive cortical irregularity.[10]

PATHOGENESIS

Since the distal femur is the site of numerous developmental variations, it is consistent that the described lesion is usually located along the posterior aspect of the

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bone is consequently weakened, and the

medial ridge of

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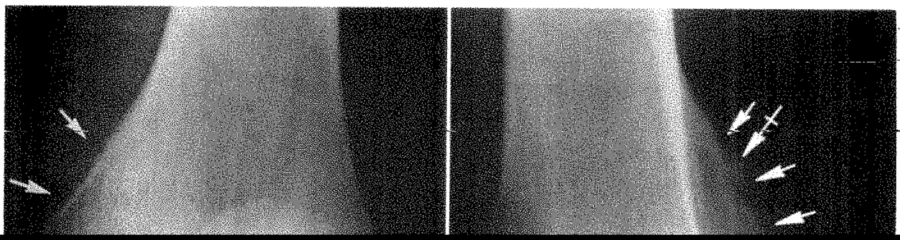

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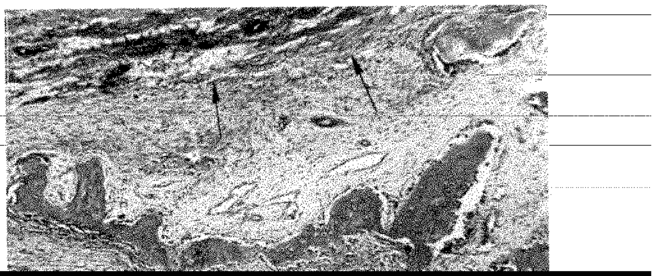
surface of the underlying bone indicating resorption of the deeper cortical layers (Fig. 9).

Following avulsion of a small fragment of bone from the cortex of the linea aspera, an inflammatory reaction is initiated and if biopsy is performed early in development, the lesion can be mistaken for a malignant



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cases have been observed in the humerus, tibia, fibula, radius, metatarsal, metacarpal and distal phalanx. All of these lesions have been located at a site of attachment of a strong muscle.[3] The importance of the described lesion is its recognition as a benign process and the avoidance of misinterpretation as a malignant neoplasm. Amputation of the leg has been reported in a case of avulsive cortical irregularity because of confusion with malignant disease[11] and, not infrequently, biopsy is unnecessarily performed because of failure to diagnose the lesion roentgenographically.[2,11,13]

SUMMARY

The avulsive cortical irregularity is commonly observed in children and adolescents along the posteromedial aspect of the distal femoral metaphysis. The described lesion has a highly characteristic location and roentgenographic appearance with some variation produced by the developmental stage.

The inciting stimulus for the development of the lesion is believed to be the intense mechanical stress in this area of active bone remodeling which results in repeated microavulsions of the cortex along the site of insertion of a portion of the adductor magnus muscle.

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the acromioclavicular joint is the subject of this paper. The most common disease conditions found in the acromioclavicular joint include: sprains; dislocations and subluxations; infectious arthritis; tuberculous arthritis; rheumatoid, degenerative and post-traumatic arthritis; tumors and bone pathology from hyperparathyroidism or metabolic diseases.[12]

The anatomy, signs and symptoms of acromioclavicular joint disease, the differential diagnosis from other causes of shoulder pain, and the roentgen diagnosis, are briefly described.

ANATOMY OF ACROMIOCLAVICULAR JOINT

The shoulder joint and girdle contain the glenohumeral articulation, the biceps muscle and tendon, the sternoclavicular and acromioclavicular joint, which function through the coordinated movements of the shoulder muscles as part of the arm-trunk mechanism. The acromioclavicular joint is a synovial joint of the diarthrodial type. The outer end of the clavicle is slightly oval shaped and the acromial end of the scapula is slightly cupped. Their surfaces are covered by cartilage and the capsular ligament is lined with synovia. In 50 per cent of the cases, a fibrocartilagenous disk is attached to the upper part of the capsule which partially divides the cavity.[9,10] This joint is strengthened by the superior and inferior acromioclavicular and coracoclavicular ligaments and allows the scapula to glide along the thoracic cage and accounts for the final 30° elevation of the humerus.[10] The joint space measures 1–3 mm. in width.

SIGNS AND SYMPTOMS OF ACROMIOCLAVICULAR DISEASE

Pain produced by acromioclavicular pathology may irradiate to the forearm and hand. Point tenderness is elicited, and the movements of the shoulder are limited on passive and active motion. Abduction of the arm produces pain and tenderness over the involved acromioclavicular joint and there may be localized crepitation on the superior aspect of the joint.[8]

With rheumatoid or degenerative osteoarthritis the pain is local and rarely referred. Tenderness of the acromioclavicular joint is elicited. Motion varies from good to none. Pain is exaggerated on abduction. Osteophytes may be palpable.[12,13]

With fractures, subluxations and dislocations of the acromioclavicular joint, symptoms of pain and reduced function of the shoulder and arm are present. Deformity is noted and swelling may be palpated over the acromioclavicular joint area. Post-traumatic degenerative arthritis of the acromioclavicular joint will develop in about one-half of the patients following trauma to the acromioclavicular joint.

ROENTGENOGRAPHY OF THE SHOULDER AND ACROMIOCLAVICULAR JOINT

Because of the variety of intrinsic pathologic conditions about the shoulder joint, and for known or suspected disease about the shoulder area, routine roentgenograms of the shoulder joints should be taken for all patients with shoulder pain. For those patients with referred shoulder pain, additional roentgenograms of the cervical and thoracic spine, chest and abdomen may be necessary. On the basis of the clinical history, visible signs and symptoms of specific disease processes, additional specialized roentgenographic techniques should be employed for exact localization and identification of the pathology. These procedures would include stereoscopy, laminagraphy, arthrography, angiography and special views which will best demonstrate the anatomy and pathology for all regions of the shoulder girdle and joint areas.

Both shoulders should be roentgenographed for comparison (Fig. 10, *A* and *B*). For those patients who are unable to cooperate for proper positioning because of pain or limitation of motion, special maneuvers are employed to obtain the required roentgenograms. The roentgen-ray beam should be angled to variable degrees, the patient may be propped and held in position, and some of the more sophisticated

In many Radiological Clinics roentgeno-

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beam angled 15° caudad, humerus in ex-

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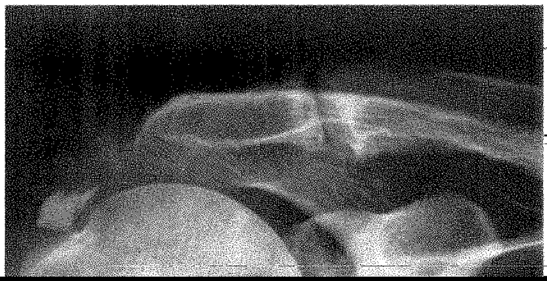

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TABLE II

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Bosworth[3] reported 2.7 per cent calcium deposits about the shoulder joints in asymptomatic persons. There is no com-

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With a high index of suspicion for pathology in the acromioclavicular articulation, and adding acromioclavicular roentgeno-

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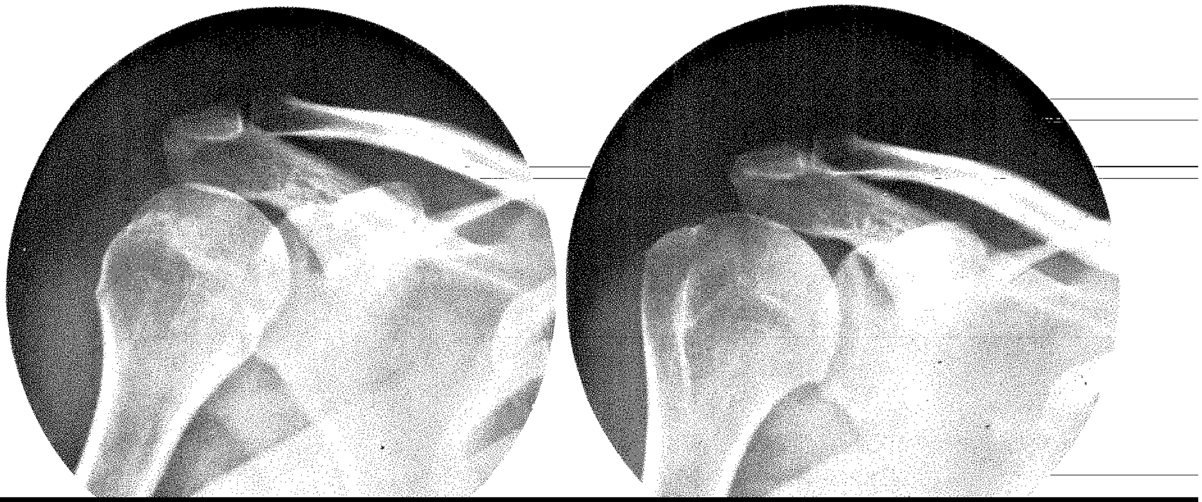

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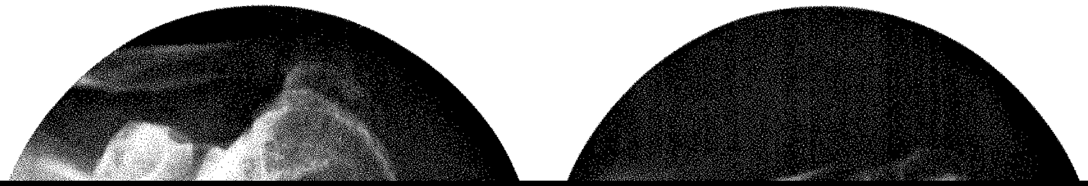

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SYNOVIAL HEMANGIOMA OF THE KNEE*

DEMONSTRATION BY ARTHROGRAPHY AND ARTERIOGRAPHY

By JOHN FORREST, M.D., *and* T. W. STAPLE, M.D.
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SYNOVIAL hemangioma was first described by Bouchut in 1856.[3] A review of the world literature by DePalma and Mauler[5] in 1964 covered 73 cases to which approximately another 30 cases have been added in the last 6 years.

The patient reported in this paper is the first in the American radiologic literature and the first reported with positive arthrographic findings.

REPORT OF A CASE

A 9 year old girl was admitted to the St. Louis Children's Hospital because of a painful right knee. Limping and decreased girth of the entire right leg had been noticed by her parents since she started to walk. No other physical or mental defects were ever noted. At the age of 5 years, pain and swelling occurred in the right knee. There was no definitive diagnosis or treatment at this time. Two months before admission, her right knee gradually became swollen, tender, painful and stiff. These symptoms worsened intermittently up to the time of admission.

Physical examination was unremarkable, except for the right leg. A tender, warm, soft, 3×4 cm. mass was present lateral to the patella. One observer thought that the mass was vascular. Full range of motion without instability was possible at the knee joint. The right leg was 1 cm. longer than the left and marked muscle atrophy was present in the right thigh and calf. The right foot was smaller than the left. The leg pulses were strong and bilaterally equal. Neurologic examination was unremarkable and no cutaneous hemangiomas were observed.

The results of a complete blood cell count and a urinalysis were normal. Aspiration of the right knee joint was attempted but only 2 cc. of blood which clotted were obtained.

Roentgenograms of the right knee showed a poorly defined soft tissue density lateral to the distal femoral epiphysis and a well defined soft tissue density in the area of the suprapatellar bursa. The bones were unremarkable and no phleboliths were seen (Fig. 1, *A* and *B*).

An arthrogram of the right knee was performed using 3 cc. of 76 per cent meglumine diatrizoate and 15 cc. of air. A lobulated 2 cm. mass was attached to the synovium near the apex of the suprapatellar bursa. The soft tissue mass lateral to the distal femoral epiphysis was extraarticular (Fig. 2, *A–C*).

The capillary phase of a right femoral arteriography demonstrated a network of tortuous, irregular, small vessels at the sites of the lateral and suprapatellar soft tissue masses. Other abnormal vessels also coursed behind the knee (Fig. 3, *A* and *B*).

At operation, a 3×3 cm. villous mass arising from the synovium was removed from the suprapatellar bursa. Another 3×3 cm. mass of the same appearance was excised from the external surface of the synovium laterally. The synovium was diffusely blood stained.

Histologically, both masses were venous hemangiomas.

DISCUSSION

Synovial hemangioma is an uncommon tumor. Of 93 hemangiomas of the extremities surgically treated at the Mayo Clinic, 6 were at the knee joint. None of the others involved a joint.[10] A few cases of involvement of other joints, especially the elbow, have been reported by other authors but the large majority has involved the knee.[5,7]

Hemangiomas of the joint may be synovial, juxtaarticular or intermediate, where both synovial and juxtaarticular components are present. The intermediate form is often a diffuse condition which is surgically unresectable.[2,9] Radiation therapy may be used in treatment of the diffuse variety.[2,6,8] Stout and Lattes[14] have divided heman-

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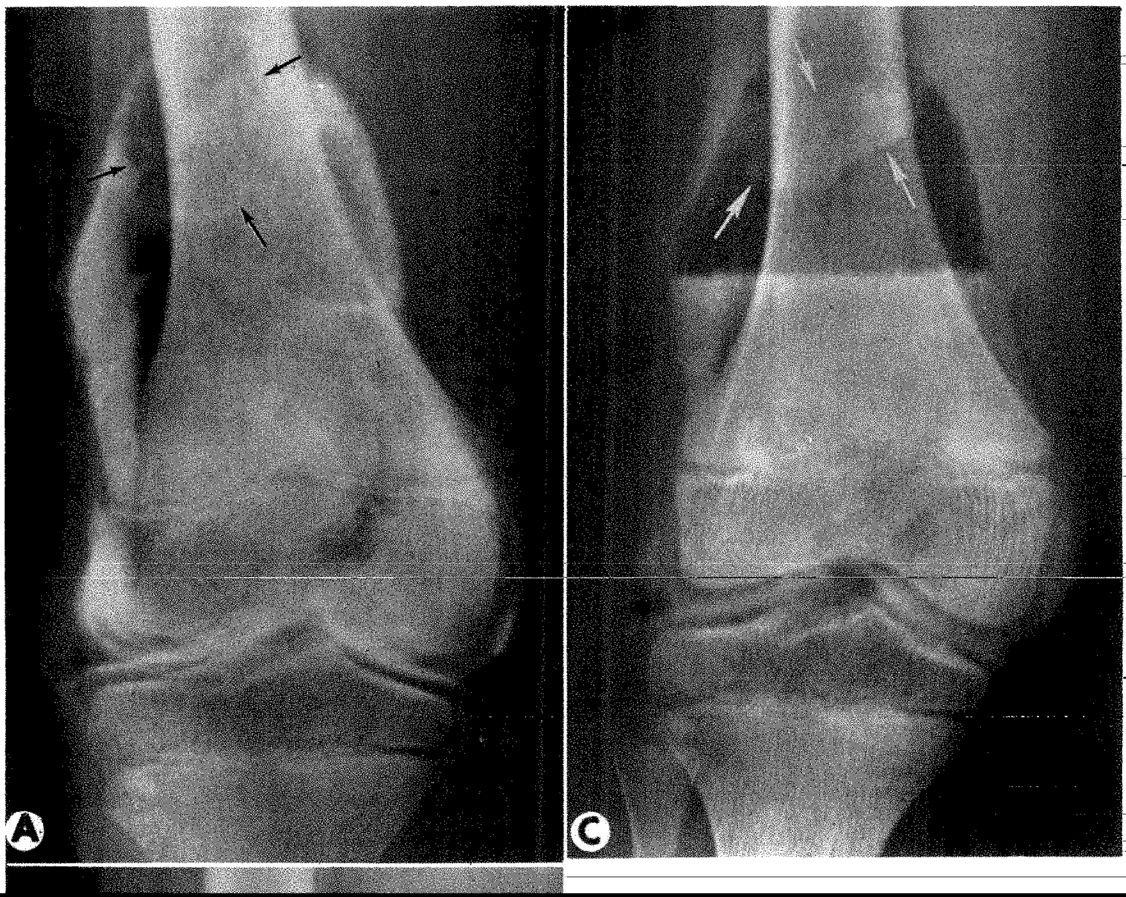

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arthrographic, and arteriographic findings is presented.

Awareness of the condition and its presentation usually will allow the diagnosis to be made or suggested preoperatively.

Arthrography and arteriography help define the extent of the tumor and the presence of multiple lesions.

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THE ADULT SYMPHYSIS PUBIS: NORMAL AND ABNORMAL*

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THE pubic symphysis is seen in a variety of roentgenographic studies. Certain clinical entities, both local and systemic, produce changes in this symphysis that may be of help in diagnosing a disease or indicating the need for further studies.

It is our purpose to present the anatomy of the normal adult symphysis pubis and to demonstrate a number of changes from which a differential diagnosis can be formulated. Although these changes have been reported previously as isolated entities, it was thought that a more inclusive discussion would be helpful.

MATERIAL AND METHOD

Fifteen specimens of the symphysis pubis obtained post mortem were studied grossly and roentgenographically. Some of these specimens were also examined microscopically.

Roentgenograms of the pelvis of 200 females and 200 males that were obtained at random from our files were studied. To avoid the difficulties that are related to age differences, only those cases whose secondary centers of ossification had fused were included in the study. Most of these roentgenograms had originally been taken as part of intravenous pyelography, in metastatic series, or after trauma.

The following roentgenographic observations of the symphysis pubis were made:

1. The greatest transverse width in the mid third
2. The relationship of the superior and inferior margins of the pubic bones in the horizontal plane
3. The contour of the adjacent pubic bones
4. The nature of the bone cortex adjacent to the symphysis.

Illustrative examples of variations and pathologic processes were selected from our files.

RESULTS

POSTMORTEM SPECIMENS

The cortices of the pubic bones of the symphysis were clearly visible and near parallel on all roentgenograms of the specimens. The transverse width of the symphysis increases from posterior to anterior. Anteriorly the ligaments are more abundant than posteriorly (Fig. 1*A*).

The symphysis is made up of hyaline and fibrocartilage which does not visibly interdigitate with the adjacent bones. This cartilage becomes less hyaline and more fibrotic toward the mid line (Fig. 1, *B* and *C*). In all specimens there was a visible mid line raphe where the cartilage became yellowish-tan in color (Fig. 1*A*). A slit or cleft readily visible when stress is applied to the pubic bones, separating them posteriorly, is commonly present. Both the raphe and the slit are best seen posteriorly. This cleft has no visible lining membrane (Fig. 1*C*).

ROENTGENOGRAPHIC OBSERVATIONS (TABLE I)

Transverse Width. The mean transverse width was 5.9 ± 1.3 mm. in males and 4.9 ± 1.1 mm. in females. Since the width of the symphysis increases from posterior to anterior, these measurements actually represent the width of the joint anteriorly.

Superior and Inferior Margin Relationship. The upper margins of the pubic bones at the symphysis pubis were on the same

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* Presented as a Scientific Essay and Exhibit at the Seventy-first Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 29–October 2, 1970.

From the Department of Radiology, Vanderbilt University and Veterans Administration Hospital, Nashville, Tennessee.

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and lower margins differed. In this latter

TABLE I

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Table II

ETIOLOGIC CLASSIFICATION

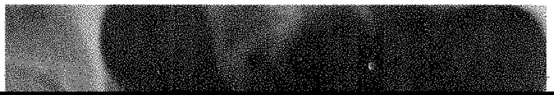
- I. Congenital
    - A. Extrophy of bladder and variations
    - B. Cleidocranial dysostosis
- II. Normal Variations
    - A. Air vacuum—nonpregnant—unusual
      pregnant—common
    - B. Increased width—pregnancy
    - C. Irregular sclerosis—most related to pregnancy
- III. Trauma
    - A. Increased width
    - B. Offset of pubic bones
- IV. Metabolic
    - A. Erosion
        - Hyperparathyroidism
    - B. Narrowing
        - Ochronosis
    - C. Calcification
        - Hyperparathyroidism
        - Idiopathic hemochromatosis
        - Idiopathic chondrocalcinosis
        - Pseudogout syndrome
- V. Inflammatory
    - A. Local
        - 1. Specific
            - Tuberculosis, *Staphylococcus* infection,

symphysis pubis. Some of these are illustrated and briefly discussed.

CONGENITAL VARIATIONS

These variations may be of width or contour. The most striking examples of abnormal separation are related to extrophy of the bladder, epispadias, and other less common abnormalities of the urogenital and anorectal regions. These abnormalities are seen in childhood but the patient may survive into adulthood (Fig. 3*A*). Muecke and Currarino[4] found that generally the severity of the associated malformation paralleled the degree of symphysis widening.

Delay in closure of mid line bone structures from the skull to the symphysis pubis is recognized in cleidocranial dysostosis. This delayed ossification may give the appearance of a wide symphysis in children. The 53 year old male with classical cleido-



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cranial dysostosis illustrated (Fig. 3*B*)

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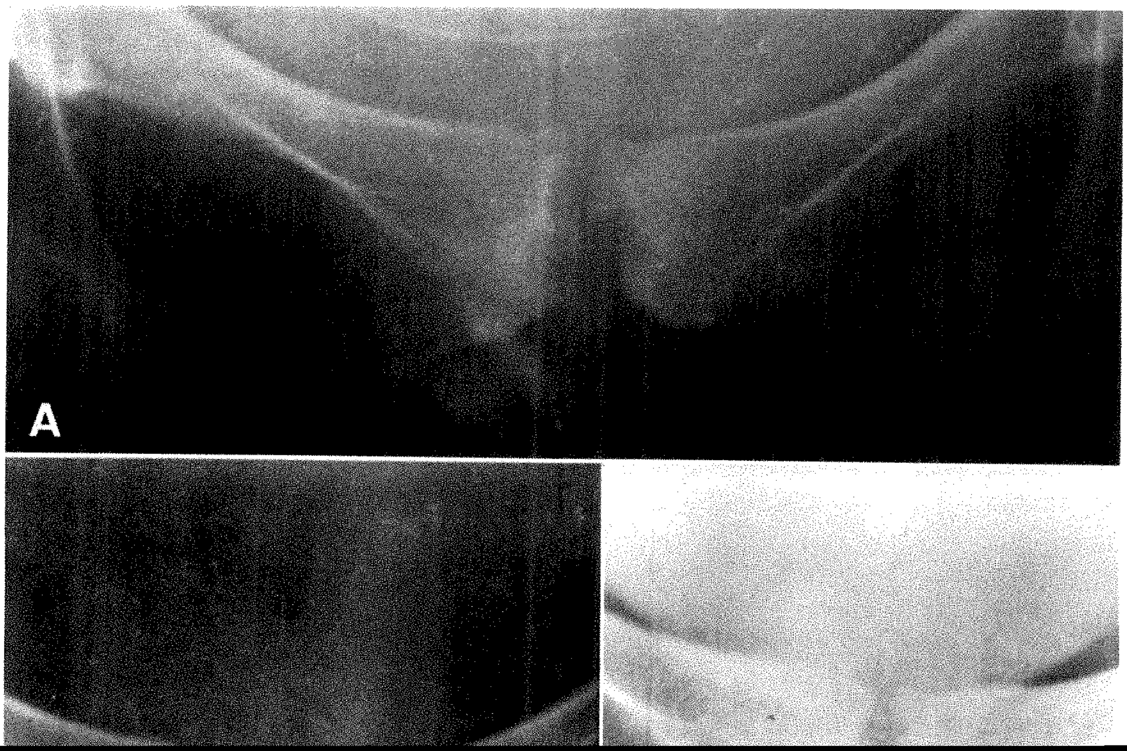
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MECHANISMS OF BONE RESORPTION IN HUMAN METAPHYSEAL REMODELING*

A ROENTGENOGRAPHIC AND HISTOLOGIC STUDY

By JOSEPH P. WHALEN, M.D.,† PATRICIA WINCHESTER, M.D.,†
LENNART KROOK, D.V.M.,§ RENATE DISCHE, M.D.,‡ *and*
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RESORPTION of bone involves two mechanisms; *i.e.*, osteoclasia and osteolysis.

Osteoclasia, first described by Kolliker, is a surface resorption brought about by the action of osteoclasts.[6] Osteolysis, on the other hand, is defined as an internal resorption of bone centered around the activity of deep-seated osteocytes.[1–5,7]

The histologic criteria of osteolysis include enlargement of osteocytes and their lacunae and metachromasia and basophilia of the surrounding matrix. Toward the end of the lytic process, which evolves in 24–36 hours—a relatively slow process compared to necrosis from trauma and other causes in which death of osteocytes occurs within 6 hours[8]—the osteocytes die. An internal resorption cavity is usually not found because of the replacement stream moving in from the apposition surface.[1,9] If, however, the osteolysis greatly exceeds apposition, internal cavities may be formed.[5]

It has been shown repeatedly that osteolysis is the primary mechanism in bone resorption, both under physiologic conditions and in diseases characterized by excessive resorption.[1–5,7] Osteoclasia is a secondary phenomenon involved in the removal of already altered bone,[2] such as occurs in necrosis from fractures or from bone altered by osteolysis.

Modeling of the metaphyseal-diaphyseal junction has generally been considered to occur through the classic mechanism of osteoclasia.

The authors present evidence that the primary mechanism of modeling in this area is osteolysis.

METHOD

Femurs of newborn infants were obtained at autopsy and roentgenograms were made on a GE 300 ma. unit, using 100 ma., 1/30 sec., 50 kvp. (Fig. 4; and 6). The femurs were then sectioned by a hand saw. Roentgenograms of the sections were obtained with the Maxitron unit employing 0.5 cm. focal spot; exposure factors were 30 kvp., 3 ma. for 2 minutes (Fig. 1).

The bone was fixed in 10 per cent neutral formalin and demineralized under water pump vacuum in 10 per cent formic acid, buffered to pH 4.5 with sodium citrate.

The decalcified bone was embedded in paraffin, sectioned at 6 microns and stained with hematoxylin and eosin and with toluidine blue.

The serial roentgenograms of the growing femurs in children who had ingested lead intermittently were reviewed.

RESULTS

ROENTGENOLOGIC EXAMINATION

In all ages the metaphyseal trabeculae were seen to converge as they reached the compact part of the shaft. Even the trabeculae arising from the lateral aspects of the growth plate remained intact and were seen to continue into the shaft area. There was symmetric condensation or shrinkage of the broad metaphyseal bone to the area of the shaft where the diameter was smaller and constant. No abrupt cut-off from the lat-

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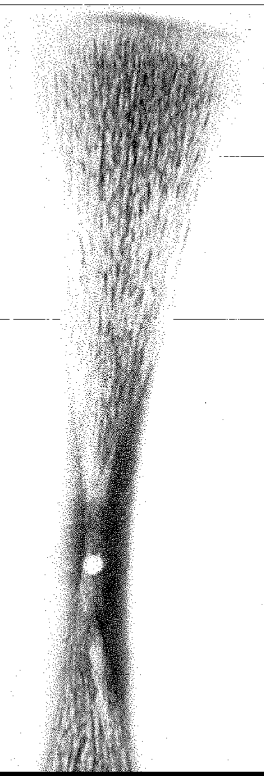
* From the Departments of Radiology† and Pathology,‡ The New York Hospital-Cornell Medical Center, New York, New York; and the Department of Pathology,§ New York State Veterinary College, Cornell University, Ithaca, New York.

Supported by the James Picker Foundation.

Fig. 1. Roentgenogram of a 0.3 cm. thick longitudinal section of the femur of a 30 week old human fetus. The trabeculae of metaphysis are shown to symmetrically converge and extend shaftward. The lateral trabeculae are not "cut."



erally placed trabeculae was detected (Fig. 2, *A* and *B*).

Lead induced marking of the growth plate was observed roentgenographically (Fig. 2*A*). The lead induced line maintained the precise original configuration as that of the growth plate, shrinking symmetrically and in proportion to the degree of modeling. Joining comparable points on the growth plate (number 1, Fig. 2*B*) and the lead induced line of prior growth plates (numbers 2 and 3, Fig. 2*B*) yielded lines corresponding to the direction of trabeculae.

HISTOLOGIC EXAMINATION

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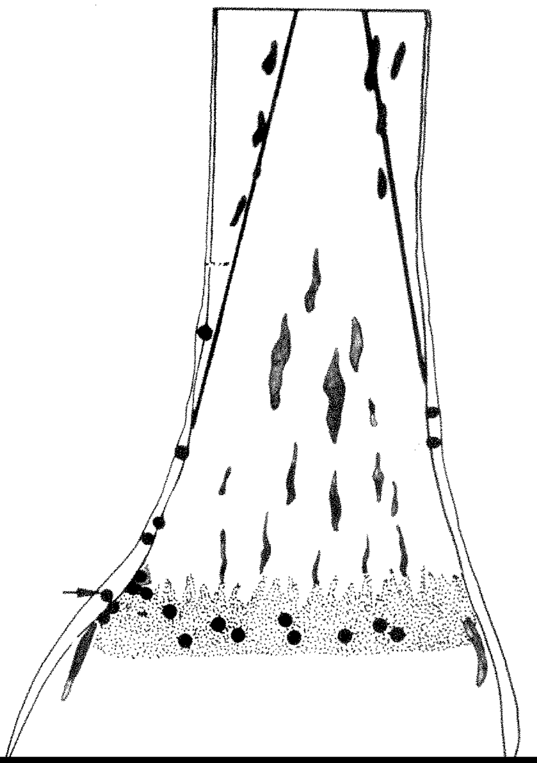
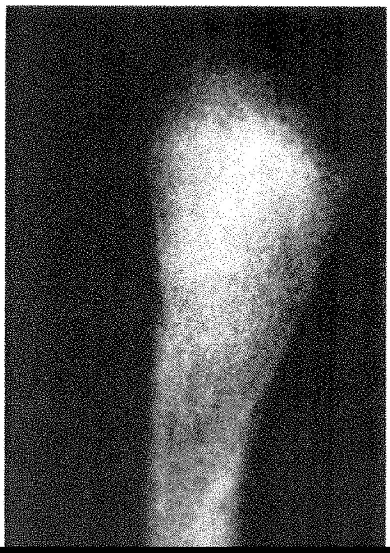
sia, these signs of osteolysis were, again, prominent in the inner third facing the medullary cavity (Fig. 8).

DISCUSSION

The symmetric conversion of the trabec-





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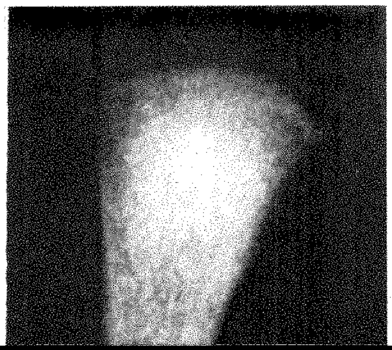

ulae formed at the growth plate, and the persistence even of those trabeculae formed from the most lateral portion of the growth plate with symmetric shrinkage, suggest diffuse resorption of bone with symmetric contraction. If a "cutting zone" of osteoclasts causes modeling, this diffuse shrinkage would not occur; likewise, the "cutting zone" would have to extend beyond what is termed the metaphyseal-diaphyseal junc-

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of the same size and the peripheral compo-

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internal resorption is responsible for the

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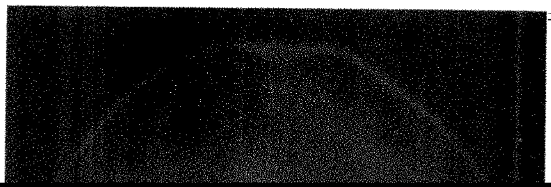

lined the family tree and the mode of transmission, which is apparently autosomal dominant with incomplete penetrance.[8] Many of these patients also have corneal leukomas, which also are heredi-

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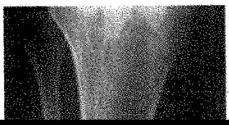
in acromegaly. Enlarged clubbed distal phalanges with the "spade finger" configuration[4] are also seen in acromegaly. In



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the squared enlargement of the phalanges are direct evidence in favor of the diagnosis of pachydermoperiostosis.

SUMMARY

Familial pachydermoperiostosis is an unusual disease which in the "forme fruste" can be confused with acromegaly.

Four cases of familial pachydermoperiostosis are presented. Although clinically similar to acromegaly, increased sellar size and increased endochondral bone growth are not seen. All the patients studied were found to have increased sinuses, cutis verticis gyrata and squaring of the phalanges. This entity should be considered by the radiologist in the differential diagnosis of acromegaly.

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TABLE I

NASOPHARYNGEAL MEASUREMENTS IN 150 MALES

	Age	Posterior Wall Thickness (p)	Roof Thickness (r)	Angle
Mean	38.8 yr.	21.0 mm.	11.5 mm.	43.7°
Standard Deviation	17.0 yr.	3.8 mm.	4.4 mm.	6.0°

TABLE II

NASOPHARYNGEAL MEASUREMENTS IN 150 FEMALES

	Age	Posterior Wall Thickness (p)	Roof Thickness (r)	Angle
Mean	41.6 yr.	19.0 mm.	8.9 mm.	42.6°
Standard Deviation	18.0 yr.	3.6 mm.	3.6 mm.	6.3°

measurement, the angle formed by lines A and B (Fig. 1), was recorded for statistical analysis.

The data were subjected to statistical analysis to determine whether there were variations in nasopharyngeal soft tissue with age and sex. In addition, because of a question as to a possible variation due to geographic location and climate, measurements were obtained on 60 adults in Hartford, Connecticut, for comparison with those obtained in Denver.

RESULTS

A summary of mean measurements and variability for both sexes is presented in Tables I and II. The effect of age upon roof thickness was significant for both sexes (p=.0001), and the mean roof thickness for males differed from that of females (p=.001). The expected roof thickness for any age in the range studied can be found from the graphs depicted in Figure 2 and Figure 3. The evaluation of posterior wall thickness was more complicated, in that

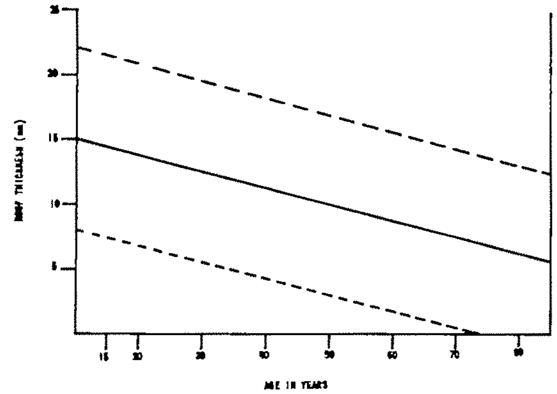


FIG. 2. Nasopharyngeal roof thickness in males plotted against age with upper and lower tolerance limits.* The regression equation is Y=16.439 −.12679X, where Y is roof thickness and X is age.

* These limits include 90 per cent of the population with 95 per cent confidence.

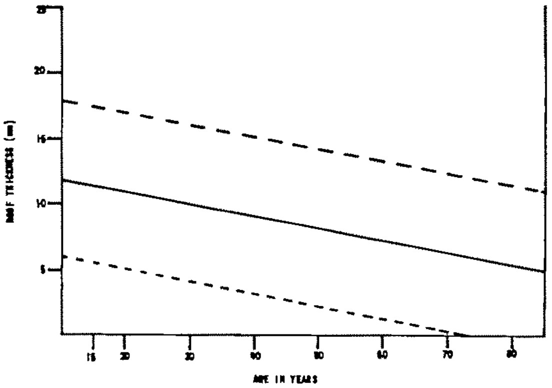
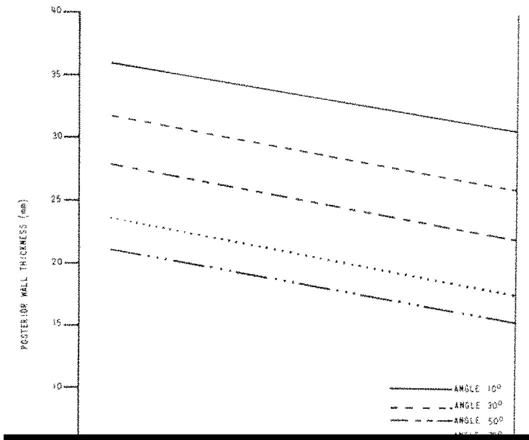


FIG. 3. Nasopharyngeal roof thickness in females plotted against age with upper and lower tolerance limits.* The regression equation is Y=12.878 −.09504X, where Y is roof thickness and X is age.

* These limits include 90 per cent of the population with 95 per cent confidence.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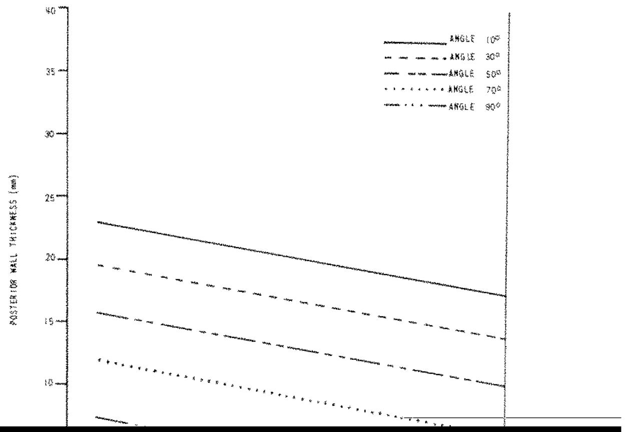
POSTERIOR WALL THICKNESS (mm)
ANGLE 10°
ANGLE 30°
ANGLE 50°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POSTERIOR WALL THICKNESS (mm)
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ANGLE 90°

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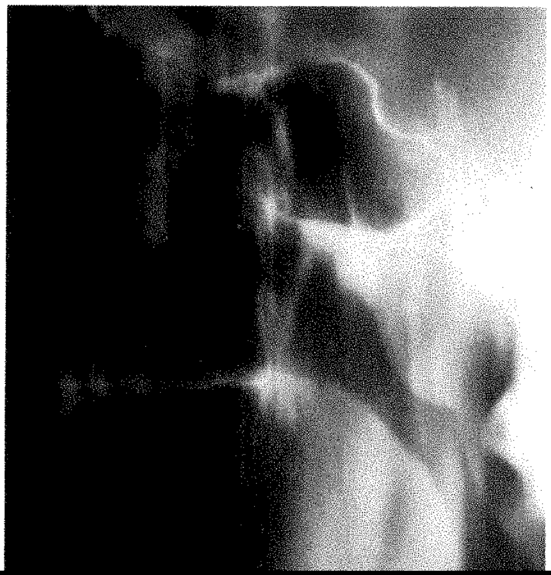

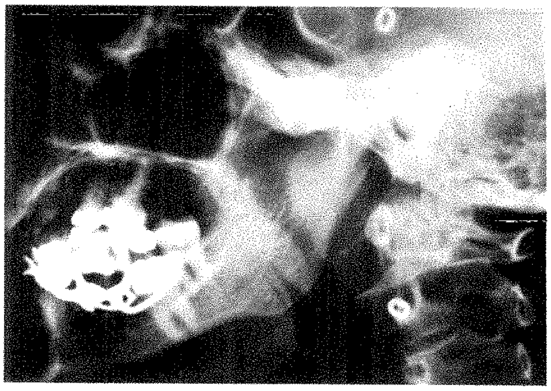

FIG. 6. Lateral skull roentgenogram of an 18 year old male with transitional cell carcinoma of nasopharynx. Note the lumpy contour of soft tissue in the nasopharynx. The measurements were within normal limits, 13 mm. for the roof and 22 mm. for the posterior wall.

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University of Colorado School of Medicine, for assistance in planning this study and for performing the statistical analysis which has made this paper possible.

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INTERCOSTAL BULGING OF THE LUNG WITHOUT EMPHYSEMA*

By KENNETH R. KATTAN, M.D., HAROLD B. SPITZ, M.D., *and* MYRON MOSKOWITZ, M.D.
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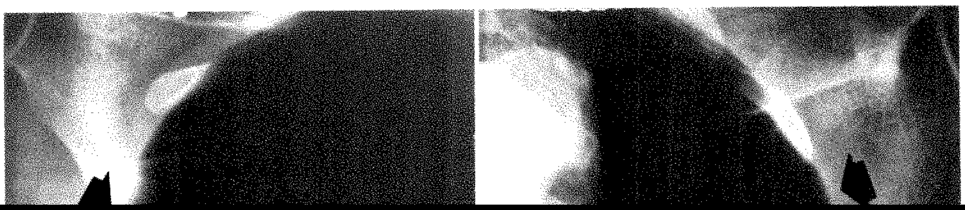
CONFIDENCE in the radiologist's ability to diagnose emphysema correctly and to correlate his observations with clinical findings, physiologic studies, and pathologic descriptions of this condition has waxed and waned through the decades.[3] One by one, the many signs once considered reliable have fallen by the wayside. Some, like Lazarus, have been resurrected. It is not our intent here to evaluate the described signs, nor even to engage in their discussion. We merely want to point out our experience with one particular roentgen sign.

Intercostal bulging was first reported in 1938 by O'Donoghue.[4] Later Schorr and Ayalon[6] described this bulging in children as indicative of emphysema. Similarly, Caffey and Silverman[1] observed that increased pulmonary tension generated by obstructive emphysema produced bulging of the lungs through the intercostal spaces in infants and small children with weak thoracic muscles. Subsequently it was pointed out that this same sign was a reliable indicator of overexpansion of the lung in adults and that it was best demonstrated in off-lateral or oblique views.[5]



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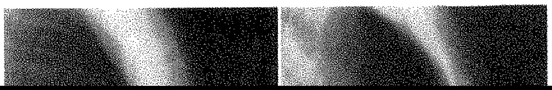
In Cunningham's Anatomy, the costal surface of the lung is described as



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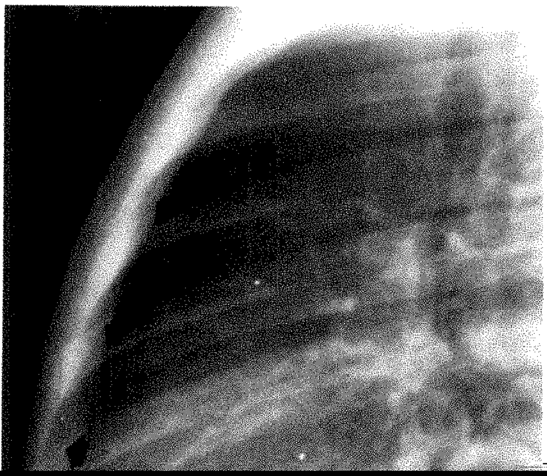

lateral projection (Fig. 4) of this thin child who had no evidence of asthma or emphysema. His medical problem revolved around rheumatic fever and was not related to intrinsic lung disease.

Case v. Autopsy on this patient demonstrated the anatomic basis for intercostal bulging. As may be noted from the accompanying photographs (Fig. 5, *A* and *B*), the external surface demonstrates ridging of the chest by the ribs. Similar matching defects are noted within the thoracic cavity. Examination at the cut surface demonstrates the marked thinning of the soft tissues between the ribs, with resultant

[redacted]

visualized in a steep oblique projection. The soft tissues of the chest wall are scalloped or indented by the lung tissue protruding into them.

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lateral roentgenograms of nonemphysematous patients, some with severe wasting and some normal.

Kenneth R. Kattan, M.D.

ROENTGENOLOGIC-PATHOLOGIC EVALUATION OF REJECTION IN ALLOGRAFTED LUNGS*

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and FRANK J. VEITH, M.D.§
BRONX, NEW YORK AND NEW YORK, NEW YORK

OBJECTIVE criteria must be established to recognize rejection and to evaluate *in vivo* the rejection process in the allografted lung. Although the pathologic anatomy of allograft rejection has been studied,[1,5] the precise pathophysiology and the locus of vascular injury have not been defined. In the present investigation allografted lungs were studied serially in untreated dogs with the following 3 purposes:

1. To describe the serial changes in the allografted canine lung by chest roentgenography, pulmonary angiography, and histologic examination.
2. To evaluate chest roentgenography and pulmonary angiography as a means of assessing the progress of rejection.
3. To attempt to clarify the pathogenesis of the vascular lesions in lung allograft rejection.

METHOD

Allotransplantation of the left lung was done in 12 mongrel dogs. The technique used in transplantation was that described by Veith and Richards,[12] although the contralateral pulmonary artery was not ligated. A standard diagnostic regimen consisting of chest roentgenography, pulmonary angiography, and biopsy of the lung, was performed at repeated intervals during the postoperative period. The animals were anesthetized with intravenous pentobarbital, their trachea was intubated and ventilation assisted when needed. Chest roentgenograms were obtained in the supine and 45° right posterior oblique (RPO) positions. A 33 inch long polyethylene catheter (inner diameter .062 inches, outer diameter .082 inches) with multiple side holes was introduced into the right jugular vein and guided under fluoroscopic control into the main pulmonary artery. Serial angiography was obtained in the right posterior oblique projection with a sequence of 2 films per second for 8 seconds as 1.75 ml. per kg. of 60 per cent methylglucamine diatrizoate was injected at a pressure of 300 pounds per square inch. In 10 of 28 studies the catheter was advanced into the lobar artery to the left lower lobe for selective angiography using 1.25 ml. per kg. of the same contrast material. Following angiographic examination a thoracotomy was performed and the left lower lobe was inspected and biopsied. The animals were not treated with immunosuppressive agents; they died between the fourth and twentieth postoperative days and were autopsied. Multiple blocks were cut from each lobe of the transplant, fixed in formalin, and stained with hematoxylin and eosin. A control series of 6 dogs was also examined with serial angiography and lung biopsy following autotransplantation of the left lung. Sixteen studies were performed at various times in the postoperative period.

Three systems were prepared for objective evaluation of the severity of rejection: (A) The first classification was based upon

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Supported in part by grants from the U. S. Public Health Service (HE 11472 and HE 11567), the Health Research Council of the City of New York, the Manning Foundation, and the John A. Hartford Foundation.

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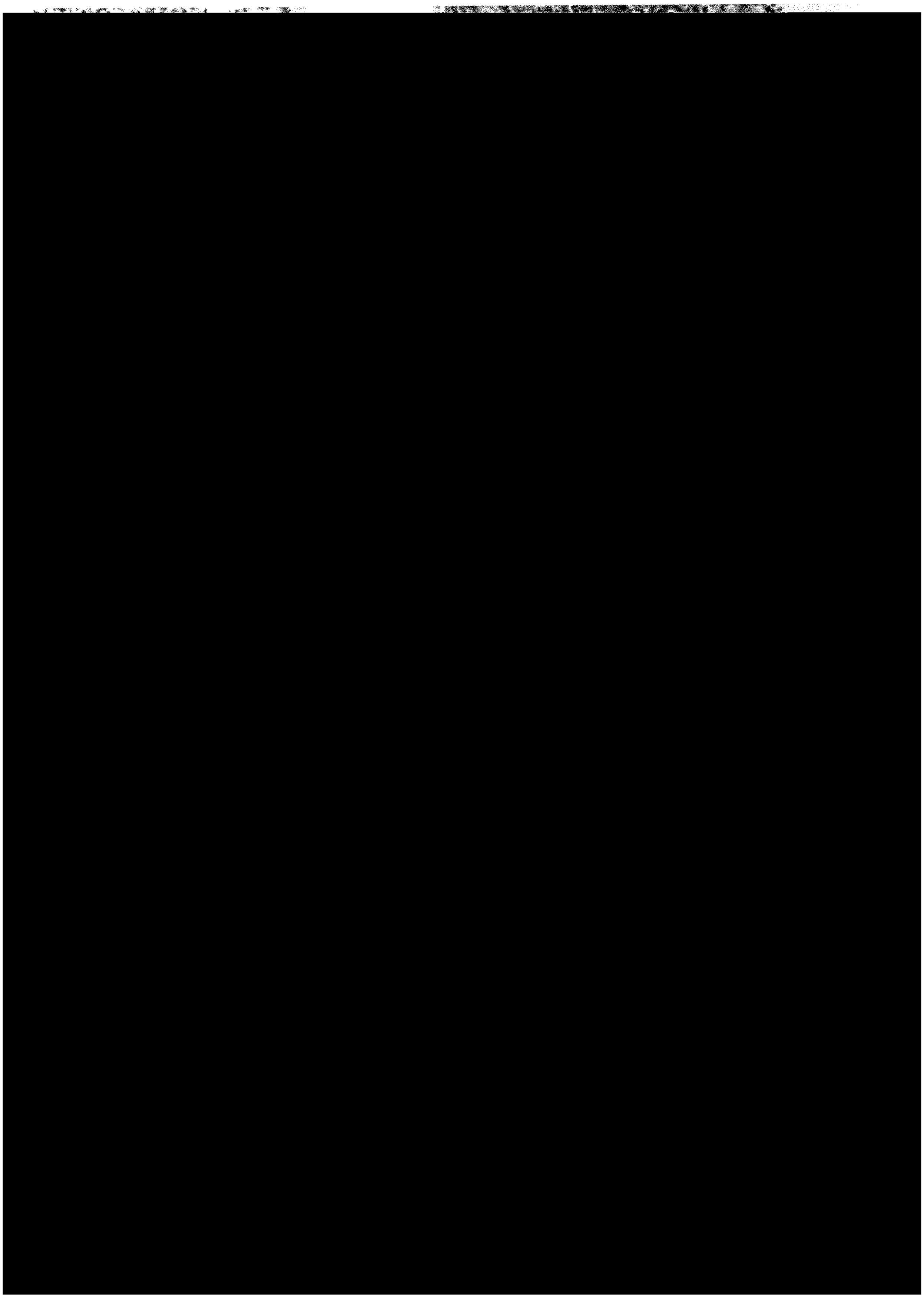

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Table II

RELATIONSHIP BETWEEN CHEST ROENTGENOGRAMS AND LUNG BIOPSY

Grade for Chest Roentgenograms	No. of Studies	Grade for Biopsy						Biopsy Lacking
		1	2	2+	3	3+	4	
N	1	1						
1	6	3						3
2	7		2	2	2			1
3	10			1	5	4		
4	4					1	3	
Total	28	4	2	3	7	5	3	4

vasculature fill promptly. There is a slowing of flow manifest first in the peripheral branches of the pulmonary artery which represents the earliest angiographic alteration. The characteristic feature of this

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branches of the pulmonary artery. Thus the zone of nonperfusion has extended centrally. Selective angiography reveals that the nonperfused tertiary and fourth order branches of the pulmonary artery are pa-

[redacted]

Table III

RELATIONSHIP BETWEEN PULMONARY ANGIOGRAMS AND LUNG BIOPSY

Grade for Pulmonary Angiograms	No. of Studies	Grade for Biopsy						Biopsy Lacking
		1	2	2+	3	3+	4	
N	6	4						2
1	4		2	1				1
2	12			2	7	2		1
3	3					2	1	
4	3					1	2	
Total	28	4	2	3	7	5	3	4

to the lung. The demonstration of an alveolar infiltrate on plain chest roentgenography in the immediate postoperative

CORRELATION OF ANGIOGRAPHY AND PATHOLOGY

Table III presents the independently determined ratings for the angiographic ex-

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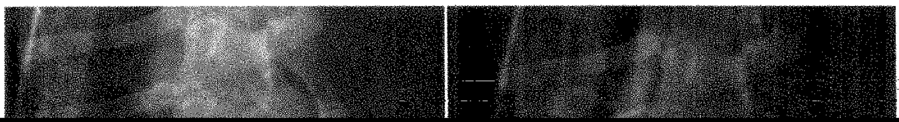

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ROENTGEN APPEARANCE OF PULMONARY VASCULARITY IN THE DIAGNOSIS OF HEART DISEASE*

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DURHAM, NORTH CAROLINA

EXCEPT for the very mild abnormalities, pulmonary vascularity almost always faithfully reflects the underlying heart disease.[1–8,10,12,15–18] In diagnosing congenital heart disease, evaluation of pulmonary vasculature has been considered one of the most important approaches.[4–8,11,12,15,17] Acquired heart diseases manifest their pulmonary vascular alterations no less accurately when the associated pathophysiology reaches a certain degree of severity.[3,6–8,10,12,16,18] The advent of modern angiography and sophisticated nuclear medicine has facilitated the evaluation of the quality (caliber and contour of vessels, distribution and pattern of blood flow, etc.) in addition to the quantity (increase or decrease of blood flow) of the pulmonary vasculature in cardiac patients.[2–8,10,12,15–20]

The present communication attempts to classify and summarize the most important roentgen signs of cardiac diseases as reflected in the pulmonary circulation. It is the authors' conviction that upon becoming familiar with the specific pulmonary vascular patterns and their physiologic significance, one should be able to easily diagnose a wide variety of cardiac diseases. For those heart abnormalities where the pulmonary vascular findings are less specific, careful study of the pulmonary vasculature will usually reveal the common denominator of a specific group of diseases.

CLASSIFICATION OF PULMONARY VASCULAR CHANGES OF CARDIAC ORIGIN

Pulmonary vascular changes of cardiac origin or pulmonary vascular abnormalities potentially affecting the heart can be classified, according to the quantity of pulmonary blood flow, into 3 basic categories. They are: (1) increased; (2) normal; and (3) decreased pulmonary vascularity. Such changes can be generalized or localized. Generalized vascular change may be associated with either normal or abnormal flow pattern, whereas localized vascular change is always accompanied by an abnormal flow pattern. By abnormal flow pattern, one may observe any one of the following: (1) cephalization (up-shifting); (2) centralization; (3) lateralization: (a) left, (b) right; and (4) localization: (a) single, (b) multiple. Increased pulmonary vascularity is customarily classified into: (1) predominantly arterial; and (2) predominantly venous in type. In case of normal or decreased pulmonary vascularity, both arteries and veins usually show a close similarity.

NORMAL PULMONARY BLOOD FLOW PATTERN

The pulmonary vascularity of a healthy person is not completely uniform throughout both lungs. Using sophisticated techniques with radioisotopes, Tauxe *et al.*[19] and Perloff *et al.*[14] found that man receives more blood in his right than his left lung. The difference is, however, small and not detectable roentgenologically. Since the pulmonary vascularity between the two lungs is practically balanced, any roentgenologically noticeable lateralization of pulmonary vascularity from one lung to the other is abnormal. Also of considerable diagnostic importance is the fact that from

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* Presented at the Seventy-first Annual Meeting of the American Roentgen Ray Society, Miami Beach, Florida, September 29–October 2, 1970.

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TABLE I

CLASSIFICATION OF CARDIAC DISEASES ACCORDING TO SPECIFIC PULMONARY BLOOD FLOW PATTERNS

A. *Increased Pulmonary Vascularity*
  1. Predominantly arterial type
    a. With normal flow pattern (flow mildly to moderately increased)
      Acyanotic; *e.g.*, left-to-right shunts, systemic arteriovenous fistula with high cardiac output and other hyperkinetic circulatory states
      Cyanotic (when great vessels are relatively normal in position); *e.g.*, Type I truncus, partial or complete transposition of great vessels without pulmonary stenosis

[redacted]

tion); *e.g.*, left ventricular failure secondary to mitral insufficiency, aortic insufficiency, myocardial disease, coronary arterial disease, systemic hypertension, left-sided obstructive lesions (including mitral stenosis, aortic stenosis, coarctation of aorta, cor triatriatum, total anomalous pulmonary venous connection below the diaphragm, pulmonary vein atresia), mediastinitis or tumor obstructing pulmonary blood vessels

    c. With abnormal flow pattern (centralization); *e.g.*, acute pulmonary edema with “bat-wings”

Table I—(*Continued*)

b. With abnormal flow pattern (cephalization); *e.g.*, any right-sided obstructive lesion with a large right-to-left shunt (decrease in pulmonary vascularity is severe and there is abundant bronchial collateral circulation—the bronchial arteries and veins tend to be more prominent in the upper medial lung fields)
c. With abnormal flow pattern (centralization); *e.g.*, severe pulmonary emphysema, diffuse pulmonary thromboembolic disease, Eisenmenger syndrome
d. With abnormal flow pattern (lateralization)
   Leftward; *e.g.*, tetralogy of Fallot with valvular pulmonary stenosis, tetralogy of Fallot with hypoplastic right pulmonary artery, tetralogy of Fallot with left-sided Blalock or Pott's anastomosis, pulmonary atresia with ventricular septal defect, patent ductus arteriosus and hypoplastic right pulmonary artery, ventricular septal defect with a pulmonary artery band
   Rightward; *e.g.*, tetralogy of Fallot with hypoplastic left pulmonary artery, tetralogy of Fallot with right-sided Blalock, Glenn, or Waterston anastomosis
e. With abnormal flow pattern (localization); *e.g.*, multiple stenoses of peripheral pulmonary arteries, multifocal and multisodic pulmonary thromboembolic disease, severe tetralogy of Fallot with abundant bronchial collateral circulation

the apex toward the base of each lung, the pulmonary blood flow becomes progressively more abundant. This phenomenon is roentgenologically discernible, and therefore well described.[3,6,8,10,12,16,18] The orderly branching of the pulmonary arterial tree from the hilum toward the periphery of the lung is apparent. The pulmonary veins are more difficult to see and they travel a completely different route, entering the left atrium at a lower level than the origin of the pulmonary artery on either side (Fig. 3).

ABNORMAL PULMONARY BLOOD FLOW PATTERN

I. CEPHALIZATION (UP-SHIFTING) OF THE PULMONARY FLOW (Fig. 8, 9)

It is generally believed that because of the erect posture of man, he receives more blood to the dependent base at the sacrifice of the apex of his lung.[8,12] This normal flow pattern may be called "caudalization" for its simplicity of tabulation in Figure 1. For reasons not completely understood, postcapillary pulmonary hypertension causes reversal of the normal pattern.[3,8,12,16,18] In other words, either anatomic or pathophysiologic obstruction to the outflow from the lung induces up-shifting of the blood with dilatation of the upper vessels and constriction of the lower ones. Again, for simplification one may call this phenomenon "cephalization" as compared with the normal "caudalization."

II. CENTRALIZATION OF THE PULMONARY FLOW (Fig. 10, 11)

By comparison with orderly branching of a normal pulmonary arterial tree, precapillary pulmonary hypertension presents a picture of central dilatation and peripheral constriction of the pulmonary vasculature. For simplification, this pattern may be called "centralization" of the pulmonary arterial flow. A similar pattern primarily involving the pulmonary venous system is exemplified by the "bat wings" appearance of severe acute pulmonary edema.[3,6,8,10,12]

III. LATERALIZATION OF THE PULMONARY FLOW (Fig. 12–15)

Although normal lungs give a right-to-left ratio of blood distribution of 52 per cent ± 3 to 48 per cent ± 3, this phenomenon is not detectable but by sophisticated radionuclide computers.[14,19] Therefore, any roentgenologically noticeable inequality of blood flow between the two lungs is considered abnormal. This phenomenon is termed "lateralization," which could be either left or right in direction depending upon the side which has the greater amount of blood. The left lateralization of pulmonary blood flow is best exemplified by valvular pulmonary stenosis. When valvular pulmonary stenosis is associated with an intact ventricular septum and without a shunt at any level, one is presented with a normal total pulmonary vascularity averaged out between the two lungs. In other words, the left lung is getting more

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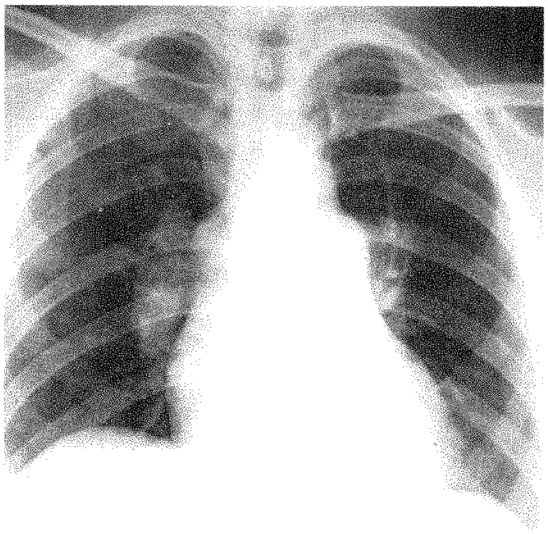


FIG. 10. Eisenmenger syndrome with a net left-to-right shunt at ventricular and atrial level. Note evidence of increased pulmonary vascularity plus a centralization flow pattern [A(1)c].

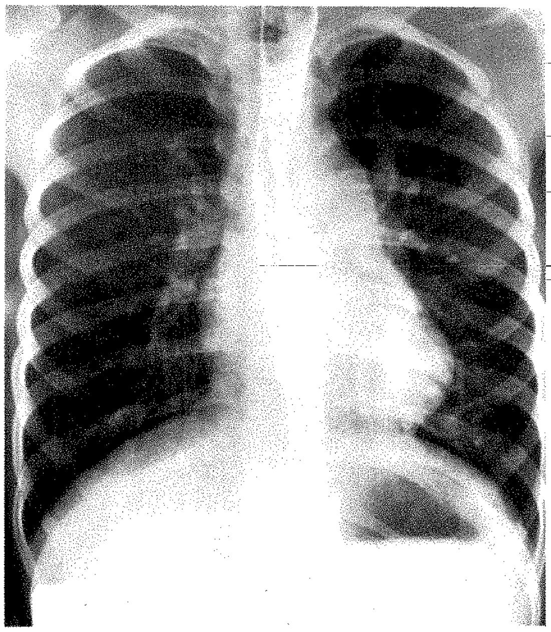
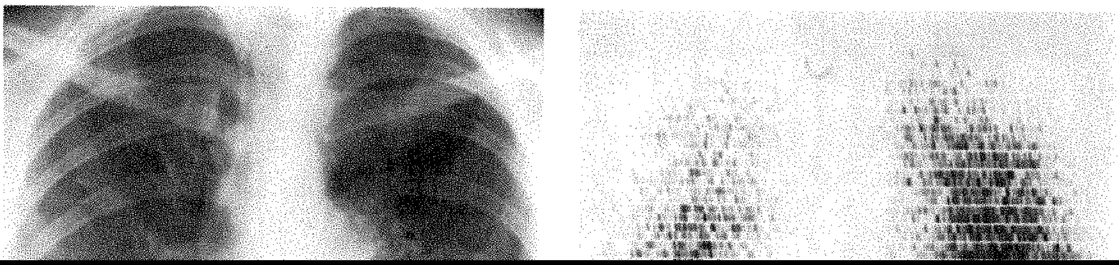


FIG. 12. Ventricular septal defect, atrial septal defect, and valvular pulmonary stenosis. Note evidence of increased vascularity plus a leftward streaming flow pattern [A(1)c].

congested, their cardiac output is usually decreased. Nevertheless, the roentgen appearance of a long standing severe mitral [redacted]

the 2 conditions is their unique flow pattern, up-shifting phenomenon in mitral [redacted]



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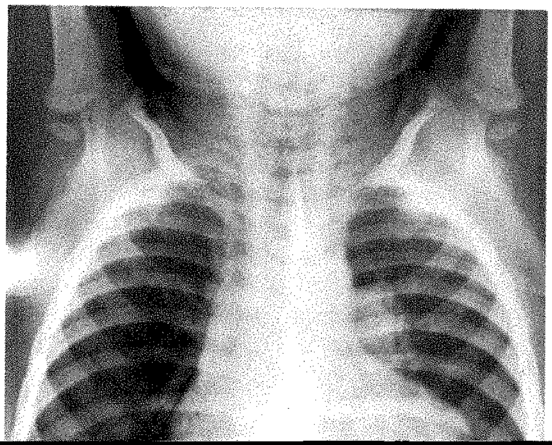

normal patterns is seen in: (1) a large left-to-right shunt with left ventricular failure (with precapillary leading to postcapillary pulmonary hypertension); and (2) a severe mitral stenosis or insufficiency (with post-capillary leading to precapillary pulmonary hypertension).

Active vascular constriction is a natural defense mechanism either to prevent a sudden surge of blood through the pulmonary circuit or to rechannel the blood from a poorly ventilated area to a normally ventilated area so as to maintain a normal

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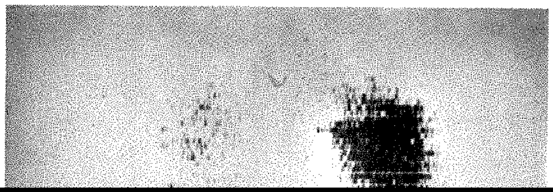
pulmonary artery and its accompanying bronchus seen on the conventional roentgenograms.

The suggested classification of pulmonary vascular changes of cardiac origin or potentially affecting the heart emphasizes



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cularity faithfully reflects the underlying heart disease.

2. Both quantity and quality of the pulmonary vascularity are determined for a more precise diagnosis.

3. A working classification of the pulmonary vascular changes of cardiac origin is suggested.

4. Principal examples of each pattern are given.

5. Certain patterns are highly specific and decidedly diagnostic of a particular disease. Others are less specific and only point to the common denominator of a group of diseases.

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AN IMPROVED METHOD OF PACEMAKER ROENTGENOGRAPHY

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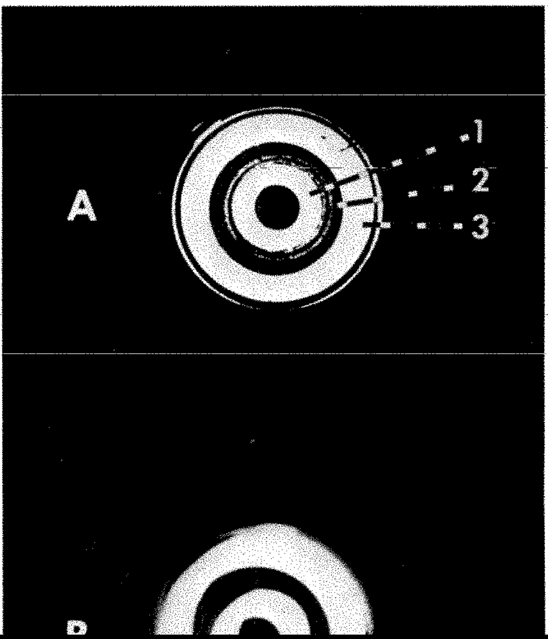
THE roentgenographic study of cardiac pacemaker batteries was first described by Greatbatch[2] and by Lillehei *et al.*[3] in 1965. Since then the usefulness of this technique in gauging their decay has been confirmed by others.[1,4,5]

GENERAL CONSIDERATION

Mercury batteries contain 2 radiopaque ring electrodes separated by radiolucent electrolyte (Fig. 1*A*). With battery discharge, metallic mercury is deposited about the inner electrode, gradually opacifying the electrolyte and finally the radiolucent center of the inner electrode (Fig. 2). In order to visualize this change, the roentgen-ray beam must be directed perpendicularly to the long axis of the cell. Because the ratio of the length of the electrodes to



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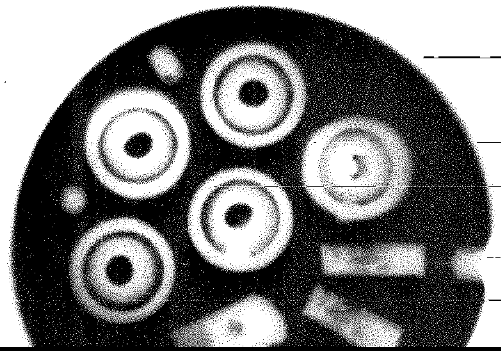

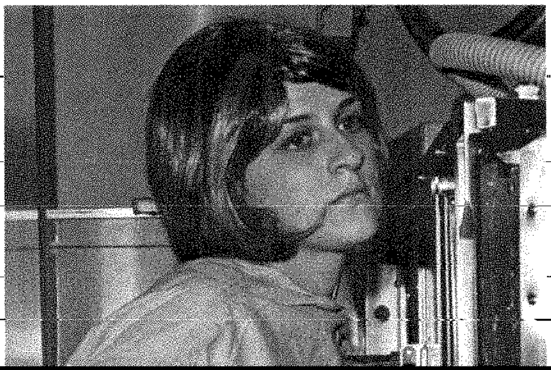

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malalignment of greater than 10°. After satisfactory positioning is confirmed by fluoroscopy, high kilovoltage, high density spot films are exposed. Abdominally placed pacemakers are seldom used because of the increased risk of electrode wire breakage. However, satisfactory spot roentgenograms are easily taken with the patient supine using the above described compression

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A new (fluoroscopic) compression cone which, when used as advised, has greatly simplified this procedure is described.

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THE ROENTGENOLOGIC DEMONSTRATION OF TINY AMOUNTS OF FREE INTRAPERITONEAL GAS: EXPERIMENTAL AND CLINICAL STUDIES*

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> *"It is impossible for any one to begin to*
> *learn what he thinks he already knows."*
> Epictetus (60–120 A.D.)

MANY years ago, while we were mem-

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patients immediately prior to the producoccurred in living patients in whom different positioning maneuvers were used. Thus, our study was designed to clarify 2 questions; *i.e.*, (1) "What is the minimum volume of intraperitoneal gas which can be demonstrated by the best radiologic technique?"; and (2) "What is the best radiologic technique for the demonstration of this minimum volume?"

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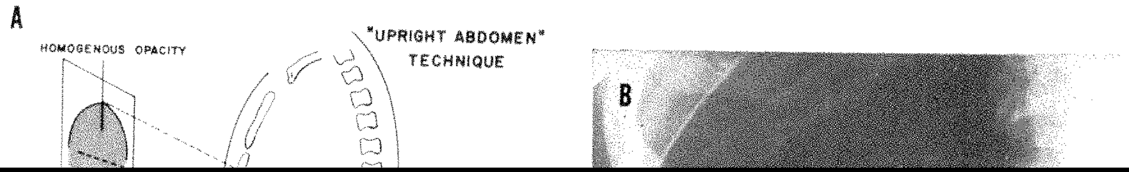
upper edge of the roentgenogram during a routine upright study of the abdomen. We centered the upright abdomen roentgenogram higher and tried to minimize the heavy exposure of the diaphragm by using a technique which necessarily resulted in underexposure of the abdominal viscera. This exposure technique was strikingly similar to that used for ordinary chest


A
HOMOGENOUS OPACITY
"UPRIGHT ABDOMEN"
TECHNIQUE
B

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A
B
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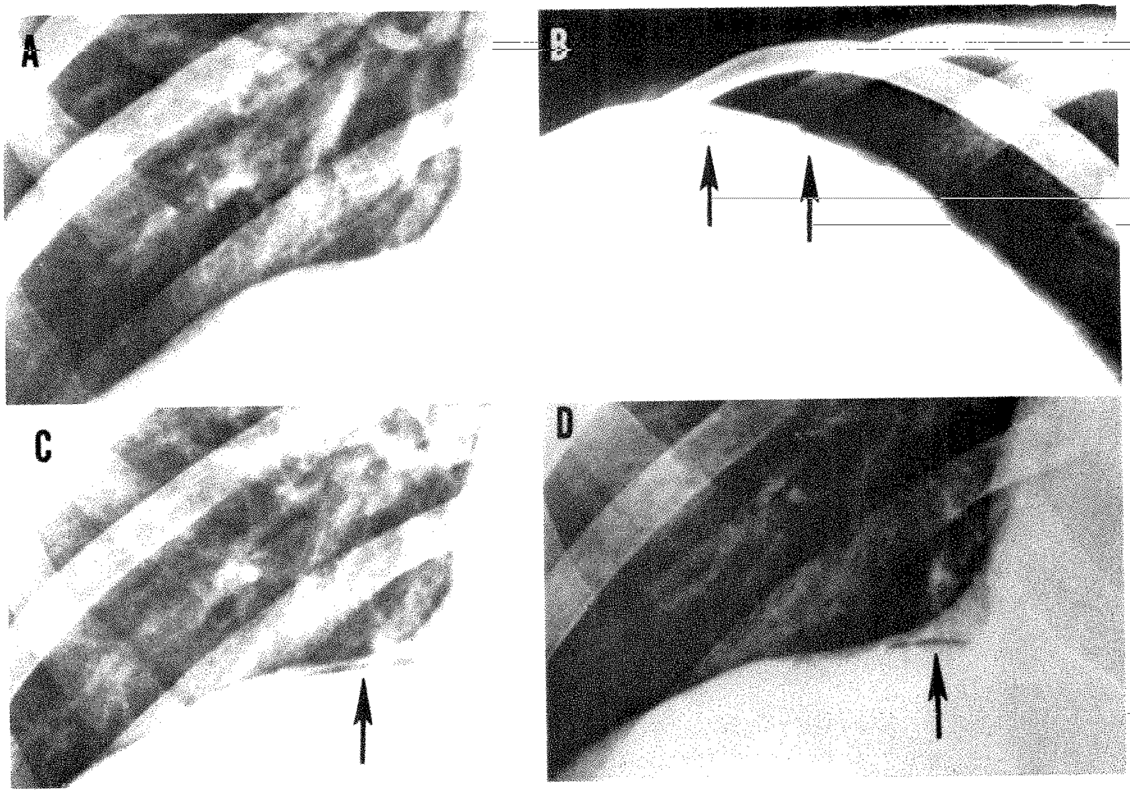

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jected could be demonstrated by either

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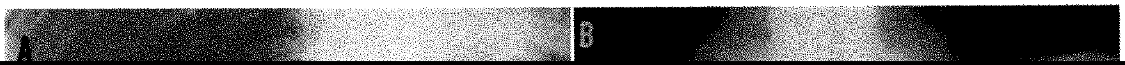
roentgenogram of the abdomen is obtained before gas has had time to reach the area between the liver and the right hemidiaphragm. If a left lateral decubitus roentgenogram is obtained, it usually is done *last* in an attempt to get more information.

It is important to perform a chest roentgenologic examination[2] in every patient being studied for an acute abdominal condition, not only because it is the most rewarding technique to use for the demonstration of tiny amounts of free peritoneal gas, but also because important clues to the etiologic mechanisms involved in the production of the acute abdominal symptomatology are often discovered in the chest. Furthermore, patients with acute abdominal conditions often have concomitant serious pulmonary or cardiac abnormalities which are often of great interest to those responsible for the surgical or medical therapeutic management.

DISCUSSION

We believe that the use of this simple technique which requires no special radiologic equipment or special preparation is the most accurate method for studying patients suspected of having a pneumoperitoneum, particularly when only tiny amounts of free peritoneal gas are present. This method has been carefully conceived, has a logical rationale, and has been eminently successful in our clinical practices without inconvenience to patients or attending physicians. It has been suggested by some that in order to favor the passage of gas from perforations of the stomach or duodenum into the peritoneum, the patient be instructed to swallow air or a small amount of carbonated beverage while lying in the left lateral decubitus position. This position favors the passage of gas into the distal half of the stomach and into the duodenum, from whence it can most easily pass through perforations into the peritoneal cavity (Fig. 5, *A–C*) without carrying liquid or solid intraluminal contents with it, as would occur if the patient were in the prone or right lateral decubitus position. Thus, in the left lateral decubitus position, peritoneal soilage will be kept to a minimum while gas can move into that part of the organ which might otherwise have very little gas available for the desired production of a pueumoperitoneum. Furthermore, it is doubtful that the left lateral decubitus position will have any deleterious effect on the patient, even if additional gas does pass into the peritoneum (Fig. 5*C*). In the event of a suspected perforation we believe it is unwise to use *any* liquid opaque media, whether it be barium or the water-soluble iodinated media, because any liquid which passes from a perforated viscus into the peritoneum can carry with it other intraluminal contents which can lead to further peritoneal contamination.

Our technique permits an efficient plain film roentgenographic study of the entire abdominal cavity within a few minutes (Fig. 8, *A–D*). We wish to emphasize that it is desirable that both the supine and erect roentgenograms of the abdomen include the lower part of the abdominal cavity (Fig. 8, *C* and *D*), which is, unfortunately, almost uniformly cut off if the upright abdominal roentgenogram is routinely centered high "to include the diaphragm." Furthermore, the chest roentgenogram, in addition to being the most efficient film for detecting tiny amounts of gas beneath the diaphragm (Fig. 8*B*, arrow), will also suffice for clarification of the status of the heart and lungs. Although we believe that the left lateral decubitus roentgenogram should be obtained *first* (Fig. 8*A*) in order to make maximum use of the time the patient has been in the left lateral decubitus position, we must emphasize our contention that this roentgenogram should not replace the erect roentgenograms in those patients who are able to stand or sit on the stretcher (Fig. 5*C*; and 9, *A–C*). This is because it is much more difficult to detect small amounts of intraperitoneal gas on the left lateral decubitus roentgenograms due to the usual heavy exposure which "burns out" tiny gas collections (Fig. 5*B*; and 9*A*). Furthermore, even in properly exposed left


A
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VALUE OF LATERAL ABDOMINAL AORTOGRAPHY*

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ALTHOUGH many angiographic laboratories have biplane roentgen ray facilities that permit roentgenography in the frontal and lateral projection during the same contrast material injection, the technique has fallen into disuse. Reference to use of lateral aortography is restricted to such specific indications as a history of abdominal angina or the demonstration of collateral vessels in the frontal angiogram.

Routine frontal and lateral roentgenograms during the initial abdominal aortography will result in a yield of information that could not be suspected clinically or detected roentgenographically. Different instances of the use of this approach to angiography are described in the following cases.

METHOD

We alternate the load of our Schönander film changers and set them to be exposed simultaneously. This has been more effective in eliminating scattered radiation than electronic alternate exposure. For the average patient the frontal exposure is set to record 1 film per second for 16 seconds. The lateral films are exposed 1 film every second for 5 seconds. The patient is centered to the frontal changer. The lateral changer is then brought as close as possible to the roentgenographic table. Resultant magnification in the lateral film is disregarded. We use an 8:1 parallel, nonfocused grid to eliminate the necessity for directing the central roentgen-ray beam to the center of the grid for the lateral changer. A 12:1 parallel, focused grid is used for the anteroposterior changer.

In the following cases the lateral projection has been of value.

REPORT OF CASES

CASE I. (Fig. 1, *A* and *B*). C. F. I., a 49 year old male, was admitted for claudication in both calfs of 6 months' duration. Angiography performed at another hospital disclosed stenosis of the distal aorta. Since runoff roentgenograms of the lower extremity had not been taken, the examination was repeated. There was no history of abdominal pain or diarrhea.

The frontal roentgenograms disclosed a lucency in the distal aorta. The true nature of this lucency could not be appreciated without the lateral projection. This view showed the origin of an arteriosclerotic plaque from the posterior wall of the distal aorta. Stenosis of the celiac and superior mesenteric arteries that was not suspected from the frontal roentgenograms was also demonstrated.

Comment. The angiogram loses some of its value if only a single projection is used. In this instance the lateral projection determined the exact nature of the distal abdominal aortic stenosis.

The detection of the visceral artery stenosis is mainly of potential advantage in the asymptomatic patient. When knowledge of visceral artery stenosis is present, subsequent symptoms of intestinal ischemia could be adequately treated prior to the occurrence of an abdominal catastrophe.

CASE II. (Fig. 2, *A* and *B*). H. R., a 62 year old man, was referred to the hospital for continuation of intraarterial chemotherapy through a catheter selectively placed in the hepatic artery. A sigmoid carcinoma had been resected and hepatic metastases documented by liver biopsy. The catheter was removed after 7 months because its lumen became occluded.

Prior to reintroduction of a second catheter, an abdominal aortogram disclosed enlargement of the gastroduodenal and pancreaticoduodenal arteries. In the lateral projection the density of contrast just proximal to celiac artery occlusion was the same as in the aorta. Distal to the occlusion the splenic artery showed lesser density of contrast because of collateral flow from the superior mesenteric artery.

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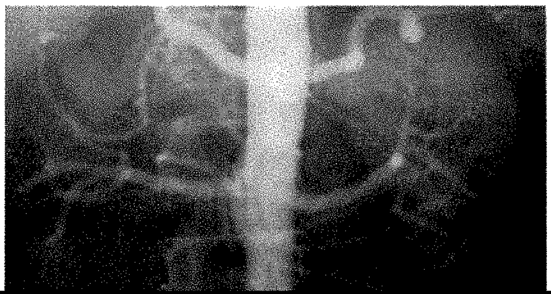
Comment. The presence of collateral vessels in the frontal view indicates either significant stenosis or complete visceral artery occlusion. The lateral aortogram in this instance documented complete celiac occlusion. Selective



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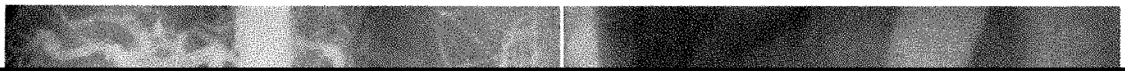
necessarily imply complete occlusion of 1 of the 3 main branches supplying the viscera. Examples of collateral vessels associated with stenosis are occasionally encountered.

An important application of the lateral aortogram is in conjunction with selective visceral angiography. The advisability and approach to selective catheterization of branch vessels of the aorta can be determined. A severely stenosed celiac or superior mesenteric artery may be completely compromised by the introduction of a selective catheter. Manipulation during selective catheterization or catheter movement during contrast medium injection may result in subintimal injection. This will diminish the size of the lumen and can result in significant compromise of the blood flow to the viscera.

The lumen of an aortic aneurysm may



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appear normal in the frontal roentgenogram. The lateral aortogram provides a second opportunity to detect the extent of clot formation.

Roentgenography in multiple projections is so basic a radiographic principle that it almost seems unnecessary to emphasize its routine use. However, since biplane aortography has been used only "where necessary" by many authorities, we feel that its advantages have been overlooked.[6] We have found it valuable in detection of completely unsuspected disease, determination of the extent of disease and in the interpretation of blood flow patterns between the visceral branches.

The documentation of the true extent of vascular occlusion will permit determination of the margin of reserve that the patient may have under certain types of pathophysiologic stress. Heart failure, shock and certain instances of bradycardia diminish blood flow to the viscera.[4,5] This effect is exaggerated with stenosis of the origin of a visceral artery.

SUMMARY

The value of the routine lateral aortogram obtained during the contrast material injection for the frontal angiogram is stressed. This application of the basic radiographic principle of recording structures or events in more than one plane has resulted in the following:

1. Detection of completely unsuspected visceral artery stenosis or occlusion. This has been of value in determining the cause of symptoms under investigation or in documenting the potential cause of subsequent disease.
2. Determination of the extent of visceral artery stenosis where its presence is already suspected.
3. Recognition of visceral artery stenosis and occlusion in the frontal aortogram. This is derived from a more complete understanding of the contrast flow pattern and the recognition of post-stenotic dilatation.
4. More complete evaluation of aortic aneurysms as to size, extent of thrombosis, associated stenotic or occlusive visceral artery changes and relationship to the visceral arteries.
5. Recognition of potentially hazardous visceral artery stenosis where selective catheterization should be avoided.

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PELVICALYCEAL EXTRAVASATIONS*

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EXTRAVASATIONS from the pelvicalyceal system, previously consid-

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preliminary roentgenogram of the abdomen. The 30 minute urogram (Fig. 1) showed indis-

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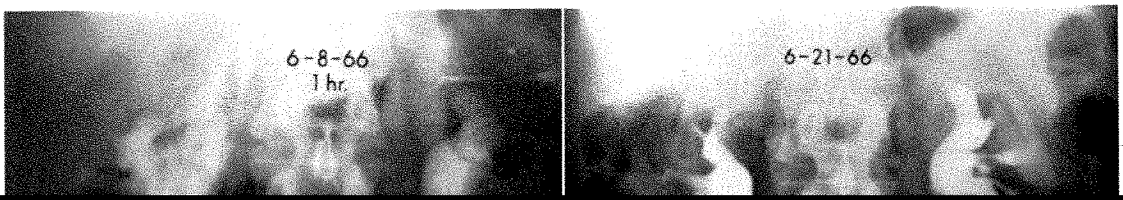

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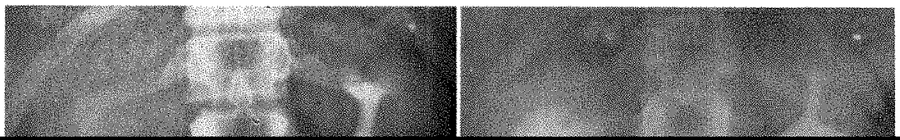

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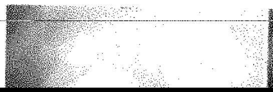
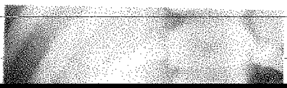
cyst was accomplished. A few days postoperatively the patient complained of left flank pain, and another urogram (Fig. 7) was ob-





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mon after ureteropelvic surgery. As long as the ureter is not obstructed, the extravasation usually terminates spontaneously. In the presence of obstruction, even of the mildest degree, extravasation will continue and an "urinoma" may develop.

DISCUSSION

The phenomenon of spontaneous extravasation, formerly considered rare, is seen with increasing frequency as techniques and contrast media improve and the radiologist's awareness increases. Numerous case reports and discussions have appeared in the literature; however, only a few have met with the strict criteria of the diagnosis of spontaneous extravasation as outlined by Schwartz *et al.*[10] These include absence of the following: (1) recent instrumentation; (2) a previous surgery; (3) external trauma; (4) a destructive kidney lesion; (5) external compression; and (6) pressure necrosis produced by a stone in the pelvis or ureter.

Renal backflow or spontaneous extravasation demonstrated during intravenous urography refers generally to the escape of contrast material outside the pelvicalyceal system. Spontaneous extravasation may be found in cases of acute and chronic obstructive uropathy, although it is more frequently observed with acute obstruction. The mechanism of spontaneous extravasation due to acute ureteral obstruction is a sudden increase in intrapelvic pressure, causing rupture of the fornix of one or more calyces.[8] The contrast medium mixed with urine then escapes into the renal sinuses and collects in the peripelvic and periureteral areas. Pyelosinus extravasation is the most common type of spontaneous extravasation.

The mechanism of pyelovenous backflow, the least common type of spontaneous extravasation, has been demonstrated by Green *et al.*[3] Using microradiologic techniques and cadaver kidneys, they concluded that pyelovenous backflow takes place through rupture of a fornix. The hydronephrotic kidney allows pyelovenous backflow to occur at a much reduced pressure, when compared to a normal kidney. According to these authors, preferential pyelovenous backflow in hydronephrotic kidneys serves as a protective mechanism and may explain induced bacteremia seen in retrograde studies in patients with ureteral obstruction.

The distinction between spontaneous forniceal rupture and an acute rent of the pelvis or ureter, due to trauma, is important because the patients with spontaneous extravasation due to acute obstruction are not as ill as those with acute rent of the pelvis and ureter. Also, conservative management is usually indicated in most cases of spontaneous extravasation, while in cases of acute rent of the pelvis or ureter, surgical intervention is usually required. During intravenous urography, the ureter is usually demonstrated down to the level of obstruction in cases of spontaneous extravasation. In cases of acute rent, however, the ureter distal to the rupture is frequently not demonstrated.

The relatively benign nature of spontaneous extravasation associated with renal colic is to be differentiated from extravasation due to rupture of the kidney following trauma. In the latter, renal function may be decreased or absent. A 30 second nephrogram may show a peripheral radiolucent line in the kidney due to blood bisecting the cortex.[5] Dilatation of the upper tract is uncommon and the extravasation is predominantly subcapsular or intraparenchymal. Surgical intervention may be required, whereas it is never necessary in spontaneous extravasation. It should be noted, however, that spontaneous extravasation is not without complications. Harrow[4] described a perinephric abscess secondary to spontaneous urine extravasation associated with renal colic. Other reported complications include peripelvic urine granuloma, fibrolipomatosis, localized retroperitoneal fibrosis, and strictures of the upper ureter and calyceal infundibula.

Spontaneous extravasation associated with chronic obstructive uropathy is not common. Urine may leak into the retro-

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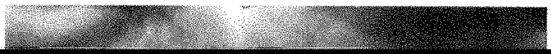
Following administration of 15 μg. of epinephrine (Fig. 2), the proximal segment of the



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cumulated reports demands continuing caution in interpreting results of postepinephrine arteriography. In fact, there is growing reason for skepticism regarding performance of this additional study following demonstration of neovascularity on the usual selective renal arteriogram.

SUMMARY

Loss of tumor neovascularity after epinephrine administration in a relatively vascular epidermoid carcinoma involving the entire kidney and adrenal is cited as further reason for skepticism regarding the use of this study to distinguish neoplasm from inflammation.

A review of the literature pertinent to epinephrine enhancement indicates that the nonspecificity of the study is well documented despite its frequent use.

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ROENTGEN FEATURES OF REFLUX INTO THE PROSTATE, SEMINAL VESICLES AND VASA DEFERENTIA*

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RELATIVELY little attention has been paid to the reflux of urine into the prostate, seminal vesicles, and vasa deferentia during the course of excretory urography. One reason for this may be the general impression that such reflux is usually confined to individuals with urethral stricture and is best demonstrated by retrograde urethrography. As a matter of fact, reflux of this type is best demonstrated by voiding cystourethrography performed at the end of intravenous urography. Moreover, in current clinical practice, many instances of such reflux result, not from a urethral stricture, but from incompetence of the internal sphincter, usually postoperative in nature.

The pathogenesis of prostatic reflux in the presence of urethral stricture associated with chronic prostatitis is quite clear. As a result of inflammatory changes in the prostate, there is loss of the normal elasticity of the duct walls and eventually fibrosis with relatively fixed dilated ductal and glandular structures. The normal pinpoint prostatic duct openings become patulous (Fig. 1). In late stages, prostatic cavities may form which communicate with the urethra.[2,4,6] When a urethral stricture is present, the increased voiding pressure in the prostatic urethra will contribute to reflux and persistence of chronic inflammatory changes. The most common etiology of chronic prostatitis with urethral stricture is still gonorrhea often complicated by secondary infection. Other infections such as tuberculosis may be responsible for similar changes.

In the absence of a stricture, if the internal sphincter is incompetent or destroyed, urine may remain in the prostatic urethra as long as the external sphincter remains contracted. Periodically, this retained urine in the prostatic urethra may reflux into ductal structures. The usual etiology for internal sphincter incompetence is surgical manipulation at the bladder neck.[1]

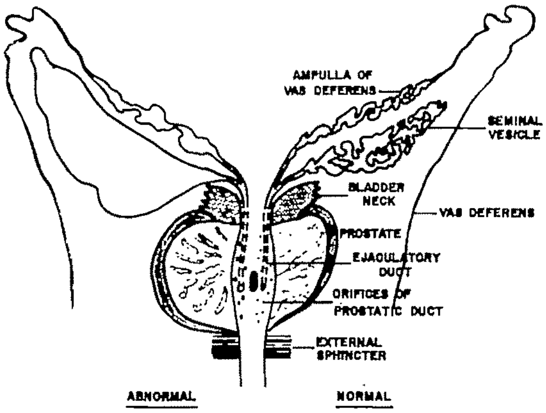

Fig. 1. Diagrammatic representation of the structures involved in reflux. The normal prostatic duct openings are pinpointed. With chronic infection and stasis, these openings become patulous. The seminal vesicle and ampullary portion of the vas deferens dilate and lose their normal convoluted appearance. The ampulla of the vas deferens may be confused with seminal vesicles if the proximal portion of the vas deferens is not visualized. The ampulla, however, is located more medially and superiorly than the seminal vesicle.

ROENTGEN FINDINGS

Reflux into the prostate may involve only several ducts (Fig. 2*A*). In other instances, the entire gland may be opacified. Although a dilated distorted duct system is the primary roentgen manifestation, one may also see diffuse opacification as the

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* Presented at the Spring Conference of the New York Roentgen Society, April 24, 1970.

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during voiding. The bladder must empty against the stricture. High pressure in the

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sulting in epididymitis. The inflammation which occurs is essentially chemical in

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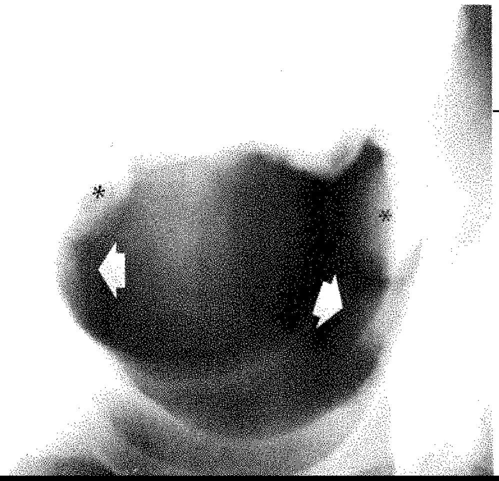

joint was believed to be an ovary. Two linear densities interpreted as round ligaments were seen originating in the reg~~ions of the internal~~ inguinal rings, extending medially and superiorly.

Exploratory laparotomy was performed, and a retroverted uterus of normal size was present in the posterior cul-de-sac, bound down by adhesions. Included in these adhesions were both ovaries and tubes. Approximately 300 ml. of old menstrual blood and debris were evacuated from the pelvis and the posterior cul-de-sac. The tubes were somewhat dilated but otherwise normal, and the ovaries were not remarkable. A Baldy-Webster round ligament suspension of the uterus was ~~performed, fol-~~

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women, but caution should be used in the diagnosis of absence of the uterus. In Case I, rudimentary uterine horns were seen (Fig. 1), permitting the diagnosis of failure of uterine development early in fetal life. But in Case II (Fig. 2), the uterus, tubes and ovaries were not seen because they were infraperitoneal, the uterus being markedly retroverted. This led us to predict, incorrectly, that the uterus was absent and that a reconstructive operation was impossible. It was only because a uterus-sized mass could be felt posteriorly on rectal examination that the roentgenologic diagnosis was questioned. At operation a normal uterus was found and a reconstructive procedure could be performed.

Unless rudimentary uterine horns can be visualized along the lateral pelvic wall, the diagnosis of congenital absence of the uterus should be made with caution by pelvic pneumography.

SUMMARY

Pelvic pneumography was misleading in 1 of 2 teen-aged girls with congenital absence of the vagina. It showed an "empty" pelvis, but a normal infraperitoneal retroverted uterus was found at operation, and a reconstructive operation could be performed.

The diagnosis of congenital absence of the uterus should be made with caution when using pelvic pneumography.

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visible and/or palpable. They considered it to be a metaplastic rather than an infiltrative process and termed it "lipoplastic" lymphadenopathy. It has been observed in lymph node areas other than the axilla, but has never been described in the pelvis. One can but speculate whether the present patient's lymphadenopathy represents an exaggerated response to the previous pelvic infection or acceleration of the normal fat replacement of the lymph node parenchyma with aging. In view of the regional nature of the process, the post-inflammatory hypothesis would appear more plausible. However, with the number of lymphangiographies performed on women with past pelvic inflammatory disease, one would anticipate other instances of such lymphadenopathy having been encountered and reported.

This pathologic process is not to be confused with the so-called pelvic lipomatosis, first described in 1959, in which large amounts of normal or chronically inflamed fat accumulate in the pelvis, distorting and displacing the pelvic viscera.[1,2] This has been described only in males, usually between the ages of 30 and 85 years, and has been shown to be vascular, unencapsulated fat that envelopes the bladder, prostate gland and rectum and is densely adherent to these structures. This lipid material is in no way confined within the pelvic lymph nodes.

It is interesting to speculate as to the causative role of the enlarged lymph nodes in creating the patient's symptoms. Their excessive size may well have contributed to the stress incontinence, and for this reason a lymph node dissection was performed as a part of the final definitive surgical procedure.

SUMMARY

An unusual instance of so-called "lipoplastic" lymphadenopathy involving the pelvic and retroperitoneal lymph nodes is reported.

The lymph node abnormality was discovered by intravenous urography and better defined by lymphangiography.

The etiology is not known, but it may represent a metaplastic process triggered by previous inflammation.

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The reddened skin had an orange-peel appearance. The clinical diagnosis was "breast abscess, rule out inflammatory carcinoma." Mammography disclosed a striking thickening of the skin, nipple retraction and an ill defined mass (Fig. 1*A*). The roentgenologic diagnosis was "probable carcinoma." The day after mammography, a biopsy was performed and 10 cc. of exudate were drained from an abscess.

Histologically the dermal manifestations featured prominent perivascular and periductal inflammation, which consisted of plasma cells and lymphocytes (Fig. 1*B*). Proliferation of vascular endothelium was also present. The lymphatic channels were not prominent.

Case II. A 49 year old woman had a recurrent abscess of the left breast. This had been incised and drained on 4 occasions over a 2 year period. Physical examination demonstrated only nipple retraction. The mammogram revealed, in addition, periareolar skin thickening, prominence of veins and ducts, a diffuse increase in density, but no mass (Fig. 2*A*).

Four days after mammography a biopsy revealed dilated dermal lymphatic channels and striking chronic perivascular inflammation (Fig. 2*B*). In addition, pronounced dermal inflammation and fibrosis were present.

FAT NECROSIS

Fat necrosis is a nonsuppurative inflammatory condition often accompanied with skin retraction, which causes it to be clinically indistinguishable from carcinoma. The disorder may follow trauma. When the lesion results in skin thickening, roentgenographic differentiation from a malignant neoplasm may be impossible.[3]

Case III. A 70 year old woman complained of a hard, fixed mass in the left breast following trauma from an automobile accident. Six months later a firm, irregular, nontender 5×8 cm. mass was palpated. There was fixation and retraction of the overlying skin. Mammography revealed an ill defined mass accompanied with thickening and retraction of skin (Fig. 3*A*). By physical examination and mammography exclusion of

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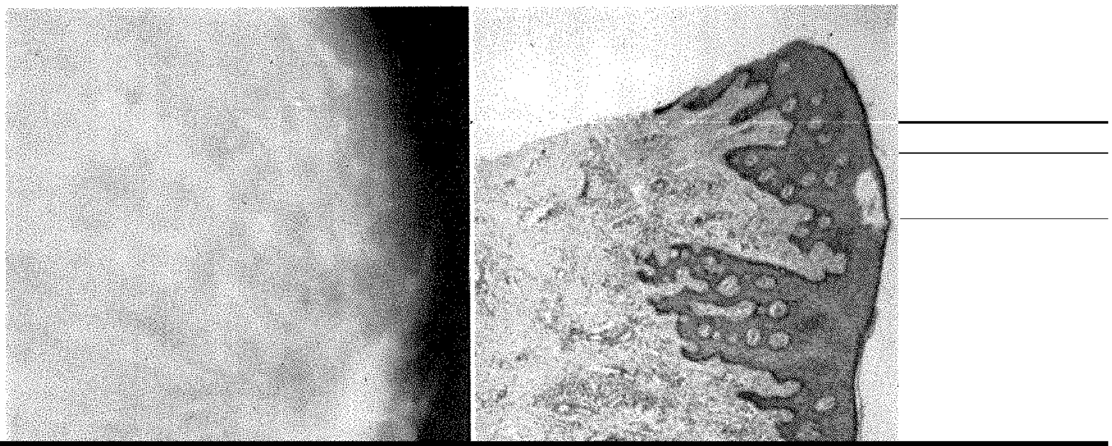

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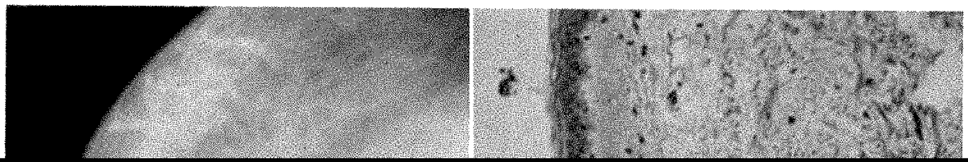

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pathologic manifestations in these disorders were nonspecific.

The single dermal pathologic feature common to all these conditions, benign and malignant, was a nonspecific, chronic inflammatory response. This response usually took the form of a perivascular, mononuclear cellular infiltration.

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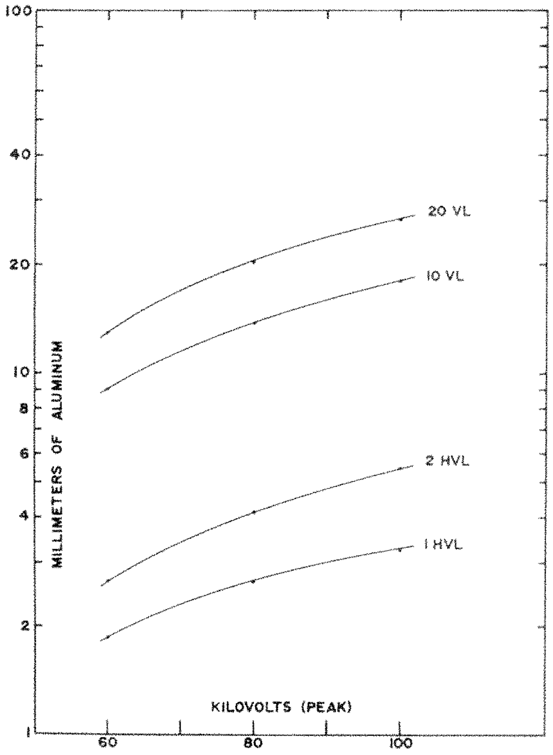

Fig. 6. Value layers for aluminum *vs.* kilovolts (peak), battery-powered equipment.

Table III

Exposure rate ratio *vs.* twentieth value layer: single-phase, three-phase, and battery-powered equipment, 3 mm. Al total filtration

20 VL mm. Al	Exposure Rate Ratio	
	Three-Phase/ Single-Phase	Battery-Powered/ Single-Phase
15	1.37	1.54
20	1.32	1.53
25	1.29	1.52
30	1.25	—
35	1.20	—

into account that considers the relative exposure rates for the 3 machines. This ratio is shown in Table III.

This study, therefore, indicates that, when a basic technique is available for any one of the 3 systems, the twentieth-value layer and exposure rate correction can be used to predict the exposure required for a given body part on other types of equipment.

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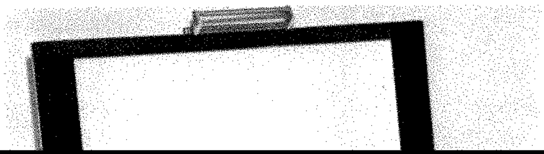

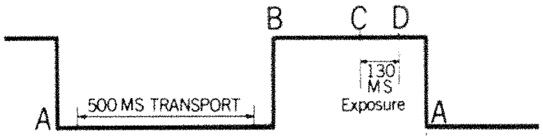

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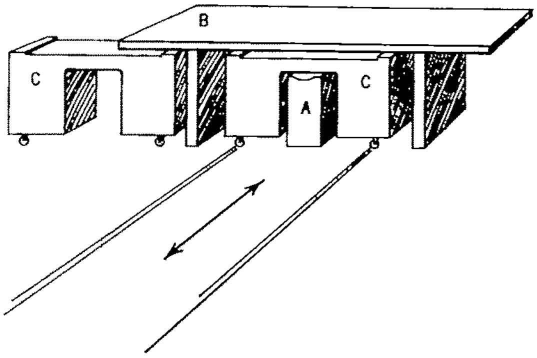

FIG. 4. Film changer (C) used in see-through fashion over image intensifier (A) or at the end of the table with a floating table top (B).

filtration to the mechanism. The high volume vacuum pump (I) produces a negative pressure of 5 pounds per square inch, thus holding both screens in contact with an atmospheric positive pressure of approximately 2,500 pounds. Intermittent evacuation of air and perfect screen film contact is possible because a relatively long time is available for closure of the intensifying screen. Perfect evacuation is accomplished by timing the roentgen-ray exposure at the end of the timing cycle as shown in Figure 3. After the compression plate has been closed at point B, the roentgen-ray exposure is initiated by an adjustable time delay relay for 130 milliseconds at the end of the compression cycle. Thus, for electronic timers, an exposure time of 1/10 second is easily available. For generators with mechanical contactors, a longer exposure time can be selected.

The changer can be used in a see-through fashion as suggested by Hoffmann and Amplatz[4] or at the end of the table with a floating table top (Fig. 4). A 14×14 and a 14×36 inch see-through changer could, therefore, be used interchangeably.

SUMMARY

A 14×36 inch roll film changer with the following advantages is described: (1) it is a see-through design and can be used underneath the catheterization table, thus allowing roentgenography and fluoroscopy without moving the patient; (2) it can also be used at the end of the catheterization table with a floating table top; (3) it has the smallest possible outside dimensions, thus representing no significant storage problem; (4) excellent film screen contact is accomplished by a single pair of intensifying screens which are closed by intermittent negative pressure; and (5) an unlimited number of programs can be selected up to a total cycle time of 30 seconds and a maximal exposure rate of 1 per second.

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Vol. 112, No. 3

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Representative on the Board of Chancellors of the American College of Radiology: Juan A. del Regato.

Fifty-fourth Annual Meeting: Boca Raton Hotel and Club, Boca Raton, Florida, May 14–19, 1972.

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* Deceased.

EDITORIALS

THE SEVENTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Seventy-second Annual Meeting of the American Roentgen Ray Society will be held at the Sheraton-Boston Hotel in the new Prudential Center in Boston, Massachusetts, September 28 through October 1, 1971. The Sheraton-Boston is most adequate to accommodate all of our activities. The exhibit space should allow all applicants to exhibit without restriction. Those that drive to the meeting will find ample parking in the Prudential Center.

This is the third time that Boston has hosted a meeting of the Society. The first was in 1913 when Henry K. Pancoast was President and the second in 1940 when Merrill C. Sosman was President. Boston is such an exciting city, steeped as it is in the history and traditions of our country. Founded in 1630, its first residents pastured their cattle on the present Boston Commons. It is the home of Harvard Medical School, M.I.T., Faneuil Hall, the Granary Burial Grounds and Durgin Park, to mention a few. Boston Commons is still the site of lively debate, and the U.S.S. Constitution is still an active commissioned ship in the U.S. Navy. There are so many things to see and do in Boston.

Registration will begin on Sunday, at 2:00 P.M. at the Sheraton-Boston Hotel in The Constitution Foyer and will be open every day thereafter from 8:00 A.M. to 5:00 P.M., except on Friday when it will close at 1:30 P.M.

On Monday, September 27th, the Annual Golf Tournament, under the Chairmanship of Henry P. Pendergrass, M.D., will be held at the Oakley Country Club in Watertown, Massachusetts. This year something new will be inaugurated. The play will begin in midmorning as a shotgun start with foursomes starting simultaneously from all tees. The players will finish at the same time in midafternoon and a late buffet luncheon will be served with the Willis F. Manges Trophy for the low net score and the Exhibitor's Trophy for the low gross score plus many other prizes being awarded at that time. The Tennis Tournament will be held under similar circumstances at the Longwood Cricket Club in Brookline, Massachusetts under the Chairmanship of Melvin Clouse, M.D. Bus transportation to the clubs and return to the Sheraton-Boston Hotel will be provided.

This change in procedure will leave Monday evening free for individual dinners, parties, theater and late supper or what you will.

The Society is indeed fortunate to have as Chairman of the Committee on Local Arrangements, Doctor Stanley Wyman, with his charming and capable wife, Jessie, as Chairman of the Ladies' Committee. Mrs. Wyman has prepared an outstanding program of entertainment for the ladies from the many, many things available in and around Boston.

On Monday:	A most unusual tour of "Boston—old and new" is planned, put on by the Ladies of the Fine Arts Museum. A box lunch is included.
On Tuesday:	A tour of the Gore Mansion in Waltham returning by bus to Anthony's

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tor of Radiology, Royal Free Hospital, London, England. He has chosen as his subject, "Obstructive Jaundice—The Radiologist, The Surgeon and The Patient." Doctor Young will be introduced by Dr. William Seaman, Professor and Chairman of the Department of Radiology, Columbia University, College of Physicians and Surgeons, New York.

The Instruction Courses will be presented on Tuesday, Wednesday and Thursday from 3:00 to 4:30 P.M. and on Friday from 1:30 to 3:00 P.M. Doctor Harold Peterson who has been the Chairman of the Committee on Instruction Courses since 1957 has again assembled an outstanding faculty who will present a wide variety of subjects for your selection. The program, with the description of the courses and who is to teach them, will be published in the August issue of the JOURNAL along with instructions for registration. Members and guests are urged to register early to make sure they can attend the courses of their choice.

On Wednesday, September 29th, at 1:30 P.M. there will be a 90 minute program titled "A Report From the American College of Radiology." Doctor Seymour Ochsner will preside.

The Annual Awards Banquet, the main social event of the week, will be held in the Grand Ballroom, on Thursday, September 30th, at 7.30 P.M. It will be preceded by a Cocktail Party in the Constitution Room at 6:30 P.M. Doctor Wyman has promised a marvelous Boston dinner, with orchestra, dancing and entertainment that will keep you interested until the very end. There will be no speeches but awards will be presented for the outstanding Scientific Exhibits and the Special Award Papers. Tickets will be on sale at the Registration Desk Monday, Tuesday, Wednesday and Thursday.

The officers of the American Roentgen Ray Society invite all Radiologists and interested physicians to attend this Seventy-second Annual Meeting. The Scientific Program and Exhibits will be most interesting. Bring your families, even if you have to take them out of school for a week, for Boston and its environs offer a lesson in American History that everyone should know.

TOM M. FULLENLOVE, M.D.
President-Elect

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the birth of two daughters, Mary Eileen and Ann Stevens. There are five grandchildren. Unfortunately, his charming wife departed this life in 1935. Barney never remarried. His homelife subsequently was spent with his sister, Florence who survives him, and his two daughters until they married.

Barney served as First Lieutenant in the Medical Corps of the Army during World War I (1917–1918). He was assigned to the Army X-ray School at Camp Greenleaf, initiating his interest in radiology. He was Consultant in Radiology at the Philadelphia Naval Hospital during World War II.

Barney was fortunate to have begun his career in radiology under the guidance of that eminent American pioneer in radiology, Dr. George E. Pfahler. He served full time as Dr. Pfahler's office and hospital assistant for ten years. This indoctrination included not only diagnostic radiology but radiotherapy. This unexcelled preceptorship was the foundation for the brilliant career that followed. Dr. Pfahler was reputed to be not only the pioneer but the leading authority on radiotherapy in America. Dr. Widmann gave the first Pfahler Lecture at the Philadelphia Medical Society in 1961.

Barney spent the greater part of his medical life in training physicians for a career in radiology. He became Assistant Professor of Radiology in the Graduate School of Medicine of the University of Pennsylvania in 1922 and rose to the rank of Professor in 1934. He was appointed Chairman of the Department (Vice-Dean) in 1945, serving in that capacity until he became Professor Emeritus in 1957. Someone has said that a teacher affects eternity; he can never tell where his influence stops (The Education of Henry Adams). Not only a great teacher, but Barney's research in his chosen field of radiation therapy was outstanding. Although not a prolific writer, approximately 100 scientific publications bear his name. Many of these were concerned with radiation in cancer therapy. His basic contributions to the treatment of skin cancer achieved world wide recognition.

From 1928 to 1960 he was Chief of the Department of Radiology at the Philadelphia General Hospital. It was at old Blockley that his principal talents in teaching, research and administration were exhibited. For many years he was Supervising Consultant in Radiology at the Fitzgerald Mercy Hospital (Darby) and Our Lady of Lourdes Hospital (Camden). Throughout his active life he was frequently called upon as Consultant in the Philadelphia Medical Schools and Hospitals in matters concerned with administration and selection of personnel in their Departments of Radiology.

The respect and admiration of his colleagues resulted in his appointment to positions of honor and responsibility in the societies of his specialty. Early in his career the Philadelphia Roentgen Ray Society elected him President (1925). This Society of his associates in Philadelphia honored him on numerous occasions. A testimonial dinner to honor Bernard Pierre Widmann was given by the Philadelphia Roentgen Ray Society at the Union League in 1961. In 1965 an oration in his honor took place in Mitchell Hall of the Philadelphia College of Physicians delivered by Professor Henry S. Kaplan of Stanford University. This was followed by a testimonial dinner at the Union League.

Barney was a member of all of the national Societies of Radiology. He served as President of the American Roentgen Ray Society at the time of their Fiftieth Golden Anniversary Meeting in St. Louis in 1950.* In the same year, he was Chairman of the Section of Radiology of the American Medical Association at their One Hundredth Anniversary meeting in Atlantic City.

One of the duties that he thoroughly enjoyed was his service on the American Board of Radiology. He was a Trustee of this Board from 1937 to 1964, the longest term of office served by any member. Barney was Chairman of the Board from 1958 to 1960. How fortunate were many young

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* *Editor's note*: The brilliant Caldwell Lecturer of that meeting was the distinguished writer of this Memoir.

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Our mutual patient contacts were of daily occurrence. During these forty years, I cannot recall any derogatory comment from patients concerning their experiences in the Widmann office; but heard innumerable remarks concerning his patience, kindness and sympathy. His advice was sought by many patients in matters unrelated to radiology because of his humane, sympathetic nature. Barney was not merely a radiologist. The designation, "radiologic physician" can best describe his professional and personal attributes.

When I think of Barney's homelife the description of the house described by Van Dyke comes to mind: "*The lintel low enough to keep out pomp and pride, the threshold high enough to turn deceit aside, the door board strong enough from robbers to defend; this door will open at a touch to welcome every friend.*"

An inscription over the portal of Barney's old hospital—the Medico-Chirugical Hospital was there for persons of the Widmann guild: "Think not the beautiful doings of thy soul shall perish; they will abide with thee forever."

A little known poem could have been written for Barney Widmann:

"*and sweetens rooms where'er he goes,*
to leave a part of him to stay."

Barney has left a part of him to stay in the hearts of his friends everywhere. A legacy all of us who knew him will cherish throughout life.

Henry L. Bockus, M.D.

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* * *

PRELIMINARY PROGRAM

SEVENTY-SECOND ANNUAL MEETING
OF
THE AMERICAN ROENTGEN RAY SOCIETY

The Seventy-second Annual Meeting of the American Roentgen Ray Society will be held at the Sheraton-Boston Hotel in Boston, Massachusetts, from Tuesday, September 28 through October 1, 1971.

The Executive Council will meet on Sunday, September 26, 1971.

In addition to the many items listed in the Editorial of this issue of the Journal, the Program Committee under the chairmanship of President-Elect, Thomas M. Fullenlove, has selected and arranged the following program for the Scientific Sessions.

Tuesday, September 28, 1971

8:30 a.m.

Call to Order, Seventy-second Annual Meeting: John F. Roach, M.D., Albany, New York, President.

Installation of President-Elect, Thomas M. Fullenlove, M.D., San Francisco, California, by John F. Roach, M.D., Albany, New York, President, and Roderick L. Tondreau, M.D., Meadowbrook, Pennsylvania, Chairman of the Executive Council.

Presentation of Past Presidents' Book to John F. Roach, M.D., by Thomas M. Fullenlove, M.D.

Presidential Address: President Thomas M. Fullenlove, M.D.

8:55 a.m.

Presiding: Thomas M. Fullenlove, M.D.
San Francisco, California

1. *Award Paper, The American Society of Neuroradiology.*
2. Evaluation of the Cerebral Circulation by Arch Aortography Supplemented by Subtraction Technique. *Jack I. Eisenman, M.D., Los Angeles, Calif., *Lawrence M. Rosen, M.D., Los Angeles, Calif., *Cecil G. Jenkins, M.D., Los Angeles, Calif., and Henry F. Pribram, M.D., Los Angeles, Calif.
3. Diagnostic Considerations in Mediastinal Emphysema: A Pathophysiologic-Radiologic Approach to Boerhaave's Syndrome and Spontaneous Pneumo-mediastinum. *F. Winston Puig, M.D., San Antonio, Texas, Lee F. Rogers, M.D., San Antonio, Texas, *Bryon N. Dooley, M.D., San Antonio, Texas, and *Leo Cuello, M.D., San Antonio, Texas.
4. The Angiographic Features of Double Aortic Arch. *W. H. Shuford, M.D., Atlanta, Ga., *R. G. Sybers, M.D., Ph.D., Atlanta, Ga., and H. Stephen Weens, M.D., Atlanta, Ga.
5. Primary Gastric Stump Carcinoma. Frieda Feldman, M.D., New York, N.Y., and William B. Seaman, M.D., New York, N.Y.
6. A Crescent Shaped Collection of Residual Telepaque in the Mid-Abdomen: A New Sign of Benign Gastric Ulcer? Sidney W. Nelson, M.D., Columbus, Ohio.
7. Unfamiliar Roentgen Findings in Pancreatic Disease. *S. Boyd Eaton, Jr., M.D., San Francisco, Calif., *Joseph T. Ferrucci, M.D., Boston, Mass., Alexander R. Margulis, M.D., San Francisco, Calif., and H. Stephen Weens, M.D., Atlanta, Ga.
8. Intestinal Ischemia. *Stover L. Smith, M.D., New Orleans, La., *Roger H. Tutton, M.D., New Orleans, La., and Seymour F. Ochsner, M.D., New Orleans, La.
9. Splenic Vein Thrombosis and Bleeding Esophageal Varices. Andrew B. Crummy, M.D., Madison, Wisc., and *Charles E. Yale, M.D., Madison, Wisc.
10. Skeletal Changes Associated with Congenital Heart Disease. *Robert I. White, M.D., Baltimore, Md., *Charles E. Jordan, M.D., Baltimore, Md., *Keith Fischer, M.D., Baltimore, Md., *Lee Lampton, M.D., Baltimore, Md., and John P. Dorst, M.D., Baltimore, Md.
11. Percutaneous Cholangiography—A Neglected Diagnostic Method. *Rex Tees-

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* By Invitation.

link, M.D., Augusta, Ga., Stephen W. Brown, M.D., Augusta, Ga., and W. H. Pool, Jr., M.D., Augusta, Ga.

12. Problems of Interpretation in Pediatric Angiography. *Albert A. Moss, M.D., San Francisco, Calif., *Robert E. Clark, M.D., San Francisco, Calif., *Alphonse J. Palubinskas, M.D., San Francisco, Calif., and *Alfred A. de Lorimier, M.D., San Francisco, Calif.
13. The First Contrast X-ray Gastrointestinal Study. Lloyd E. Hawes, M.D., Boston, Mass.

Tuesday Evening, September 28, 1971
Eight-thirty O'Clock

THE CALDWELL LECTURE

Obstructive Jaundice—The Radiologist,
The Surgeon and The Patient
By
William B. Young, M.D.

Director of Radiology, Royal Free Hospital
London, England

Introduction by William B. Seaman, M.D., Professor and Chairman, Department of Radiology, Columbia University College of Physicians and Surgeons, New York, N.Y.

Presentation of the Caldwell Medal to Dr. Young by President Fullenlove.

Wednesday, September 29, 1971

8:30 A.M.

Presiding: Russell H. Morgan, M.D.
Baltimore, Maryland

14. Pulmonary Interstitial Emphysema in the Newborn. J. Scott Dunbar, M.D., Montreal, Canada, *B. D. Fletcher, M.D., Montreal, Canada, and *E. W. Outerbridge, M.D., Montreal, Canada.
15. Long Time Study of Patients with Peyronie's Disease Treated with Irradiation. Charles L. Martin, M.D., E.E., Dallas, Texas.
16. A System for Achieving a Precise Biological Dose with Permanent Implants of Short Half-Life Radioisotopes. *Frank Ellis, M.D., Los Angeles, Calif., and *H. R. Haymond, Ph.D., Los Angeles, Calif.
17. Acute Mesenteric Infarction. *John R. Scott, M.D., Philadelphia, Pa., Wallace T. Miller, M.D., Philadelphia, Pa., *May Urso, M.D., Philadelphia, Pa., and *Robert C. Stadalnik, M.D., Davis, Calif.
18. Intestinal Pseudo-obstruction: The Known and the Unknown. *Albert A. Moss, M.D., San Francisco, Calif., *Henry I. Goldberg, M.D., San Francisco, Calif., and Alexander R. Margulis, M.D., San Francisco, Calif.
19. Vector Principle in the Differential Diagnosis of Abdominal Masses; II. Right Upper Quadrant. *Joseph P. Whalen, M.D., New York, N.Y., John A. Evans, M.D., New York, N.Y., and *Morton A. Meyers, M.D., New York, N.Y.
20. The Roentgenologic Appearance of Sclerosing Cholangitis. *Stephen R. Brown, M.D., Rochester, Minn., Harley C. Carlson, M.D., Rochester, Minn., and *Jurgen Ludwig, M.D., Rochester, Minn.
21. Angiography of Wilms' Tumor. *Robert E. Clark, M.D., San Francisco, Calif., *Albert A. Moss, M.D., San Francisco, Calif., *Alfred A. de Lorimier, M.D., San Francisco, Calif., and *Alphonse J. Palubinskas, M.D., San Francisco, Calif.
22. Evaluation of Renal Masses by Roentgenologic and Ultrasonic Methods. *Bruce D. Doust, M.B., Eloise, Mich., *Vivienne L. Doust, M.B., Eloise, Mich., *Helen C. Redman, M.D., Eloise, Mich., and Harry W. Fischer, M.D., Eloise, Mich.
23. The Value of Routine Tomography in Excretory Urography. *David F. Powell, M.D., Rochester, Minn., *Glen W. Hartman, M.D., Rochester, Minn., and David M. Witten, M.D., Birmingham, Ala.
24. Successful Clinical Radioprotection of Kidney by the Selective Infusion of Epinephrine in Two Cases. *Richard J. Steckel, M.D., Los Angeles, Calif., *Peter Tobin, M.D., Los Angeles, Calif., *James D. Collins, M.D., Los Angeles, Calif., Justin J. Stein, M.D., Los Angeles, Calif., L. R. Bennett, M.D., Los Angeles, Calif., *Donald P. Anderson, M.D., Los Angeles, Calif., *Harold Snow, D.V.M., Los Angeles, Calif., and *Morris Barenfus, D.V.M., Los Angeles, Calif.
25. Preoperative Irradiation of Renal Cell Carcinoma—A Correlation of Angiographic and Histologic Changes. *J.

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* By Invitation.

Duncan Craven, B.M., F.F.R., Los Angeles, Calif., and *James W. Lecky, M.D., Los Angeles, Calif.

26. Experimental Arteriography During Perfusion Preservation of Kidneys. Ralph J. Alfidi, M.D., Cleveland, Ohio, and *Magnus Magnusson, M.D., Cleveland, Ohio.
27. Radiology in Archaeology—A Study of the Egyptian Pharaohs. Walter M. Whitehouse, M.D., Ann Arbor, Mich., *James E. Harris, D.D.S., Ann Arbor, Mich., *Paul V. Ponitz, D.D.S., Battle Creek, Mich., *Arthur T. Storey, D.D.S., Ph.D., Toronto, Canada, *Kent Weeks, Ph.D., New York, N. Y., and *William J. Russell, R.T., Ann Arbor, Mich.

Wednesday, September 29, 1971

1:30 P.M.

Presiding: Seymour F. Ochsner, M.D.
New Orleans, Louisiana
Chairman, Board of Chancellors,
American College of Radiology

A REPORT FROM THE COLLEGE

Thursday, September 30, 1971

8:30 A.M.

Presiding: Roderick L. Tondreau, M.D.
Meadowbrook, Pennsylvania

28. *Award Paper: The Society for Pediatric Radiology*. To be announced.
29. Pulmonary Atresia with Intact Ventricular Septum—New Developments in Diagnosis and Treatment. Kent Ellis, M.D., New York, N.Y., *William Casarella, M.D., New York, N.Y., *Welton M. Gersony, M.D., New York, N.Y., and *Frederick O. Bowman, Jr., M.D., New York, N.Y.
30. Aeration Disturbances Secondary to Pulmonary Infection. Charles E. Shopfner, M.D., Kansas City, Mo.
31. Necrotizing Enterocolitis of Infancy. Rubem Pochaczevsky, M.D., Brooklyn, N.Y. and *E. George Kassner, M.D., Brooklyn, N.Y.
32. The Opaque Lung in Lobar Emphysema. *Charles J. Fagan, M.D., Galveston, Texas, and *Leonard E. Swischuk, M.D., Galveston, Texas.
33. The Metaphysis of Growing Bone: An Area of Marked Dynamic Alteration. *Patricia Winchester, M.D., New York, N.Y., *Renate Dische, M.D., New York, N.Y., *Joseph P. Whalen, M.D., New York, N.Y., *Lennart Krook, D.V.M., New York, N.Y., *Eladio Nunez, Ph.D., New York, N.Y., and Neil O'Donohue, M.D., New York, N.Y.
34. Cerebrospinal Fluid Imaging: A System of Classification of Hydrocephalus Based upon Correlation of Cisternograms with Clinical Manifestations and Pneumoencephalograms. *A. Everette James, Jr., M.D., Sc.M., Baltimore, Md., *Paul F. J. New, M.D., Baltimore, Md., *Fred J. Hodges, III, M.D., Baltimore, Md., *Harold J. DeBlanc, Jr., M.D., Baltimore, Md., *John C. Harbert, M.D., Washington, D. C., and *Henry N. Wagner, Jr., M.D., Baltimore, Md.
35. Lung Scan Patterns in Pulmonary Embolism *Versus* Those in Emphysema and Congestive Heart Failure. *David L. Gilday, M.D., Toronto, Canada, and *A. Everette James, Jr., M.D., Sc.M., Baltimore, Md.
36. Radioisotope Angiography Using Xenon 133, Technetium 99m and Indium 133. *Vladimir B. Bosnjakovic, M.D., Ph.D., Los Angeles, Calif., and Leslie R. Bennett, M.D., Los Angeles, Calif.
37. Radiographic Display of Isodose Curves from Radium Sources and External Beams Utilizing Sabattier Effect. *Frank C. Wilson, M.D., San Antonio, Texas, *Robert G. Waggener, Ph.D., San Antonio, Texas, Lee F. Rogers, M.D., San Antonio, Texas, and Peter Zanca, M.D., San Antonio, Texas.
38. 2 MV Sector Scanning Irradiation in Breast Cancer—Technique and 15 Year Clinical Experience. Ferdinand A. Salzman, M.D., Boston, Mass., Magnus I. Smedal, M.D., Boston, Mass., John G. Trump, Sc.D., Boston, Mass., *Kenneth A. Wright, M.S., Boston, Mass., and *Herbert W. Mower, B.S., M.S., E.E., Boston, Mass.
39. Solitary Plasmacytoma of the Upper Air and Food Passages. *Lawrence W. Kotner, M.D., Boston, Mass., and C. C. Wang, M.D., Boston, Mass.
40. Severe Congenital Heart Disease in the Neonatal Period: A Functional Approach

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* By Invitation.

to Emergency Diagnosis. *Michael T. Gyepes, M.D., Los Angeles, Calif., and *William Vincent, M.D., Los Angeles, Calif.

41. A Radiologic Approach to the Differential Diagnosis of Surgical and Non-Surgical Jaundice. *Alan G. Greene, M.D., Boston, Mass., and *Norman L. Sadowsky, M.D., Boston, Mass.

Friday, October 1, 1971

8:30 A.M.

Presiding: William B. Seaman, M.D.
New York, New York

42. Current Status of Sialography. *Heun Y. Yune, M.D., Nashville, Tenn., and Eugene C. Klatte, M.D., Nashville, Tenn.
43. The Roentgenographic Appearances of Metastatic Malignant Pheochromocytoma. *Reese Epperson James, M.D., Rochester, Minn., Hillier Locke Baker, Jr., M.D., Rochester, Minn., and Paul William Scanlon, M.D., Rochester, Minn.
44. Mammographic Indications for Biopsy of Clinically Normal Breasts; Correlation with Pathological Findings in Seventy-two Cases. James V. Rogers, Jr., M.D., Atlanta, Ga., and *R. Waldo Powell, M.D., Atlanta, Ga.
45. Breast Thermography after Four Years and 10,000 Studies. Harold J. Isard, M.D., Philadelphia, Pa., *Warren Becker, M.D., Philadelphia, Pa., *Ruth Shilo, M.D., Philadelphia, Pa., and *Bernard J. Ostrum, M.D., Philadelphia, Pa.
46. Pseudo Tumors of the Brain: Abnormal Vascular Patterns in Benign Intracranial Lesions. *Norman E. Leeds, M.D., Bronx, N.Y., and *Herbert I. Goldberg, M.D., Philadelphia, Pa.
47. Deep Venous Drainage into the Vein of Galen: Selective Injection and Illustrated Cases. *John Crockett, M.D., Detroit, Mich., and Harvey I. Wilner, M.D., Detroit, Mich.
48. Splenic Trauma: Assessment of Problems in Diagnosis. *Robert E. Gold, M.D., Eloise, Mich., and *Helen C. Redman, M.D., Eloise, Mich.
49. Roentgenographic Diagnosis in Blunt Abdominal Trauma with Retroperitoneal Duodenal Rupture. *Margaret E. Toxopeus, M.D., Detroit, Mich., *Charles Lucas, M.D., Detroit, Mich., and Kenneth L. Krabbenhoft, M.D., Detroit, Mich.
50. Angiographic Pattern of the Primary Retroperitoneal Tumors; The Role of the Lumbar Arteries in Evaluation of These Masses. Robert M. Lowman, M.D., New Haven, Conn., *Dorothea R. Peck, M.D., New Haven, Conn., and *Dara Dubash, M.D., New Haven, Conn.
51. Colonofiberoscope—A New and Valuable Diagnostic Modality. *Hiromi Shinya, M.D., New York, N.Y., *William I. Wolff, M.D., New York, N.Y., Abraham Geffen, M.D., New York, N.Y., and *Sezan Ozoktay, M.D., New York, N.Y.
52. Percutaneous Transhepatic Cholangiography: The Differential Diagnosis of Bile Duct Pathology. *Martin P. Fleming, M.B., B.Ch., Rochester, Minn., Harley C. Carlson, M.D., Rochester, Minn., and *Martin A. Adson, M.D., Rochester, Minn.
53. Superior Vena Caval Obstruction in Histoplasmosis. *Jonathan Thomas Fairbank, M.D., Burlington, Vt., *John Peter Tampas, M.D., Burlington, Vt., and *George Longstretch, M.D., Burlington, Vt.
54. *Award Paper, The American Radium Society:* Recent Advances in the Management of Hodgkin's Disease. *Henry S. Kaplan, M.D., Stanford, Calif.
55. Scattered Radiation from Bedside Radiographic Examinations. E. Dale Trout, D.Sc., Corvallis, Ore., *John P. Kelley, B.S., Corvallis, Ore., and *Jo Ellen Kellner, M.S., Corvallis, Ore.

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* By Invitation.

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NEWS ITEMS

FIRST ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY

As indicated in the February 1971 issue of the Journal (p. 425), although the International Society of Radiology has not been concerned directly with the promotion of zonal activities, two large regional groups, the European Association of Radiology and the Inter-American Congress of Radiology, conduct regional congresses at four yearly intervals about midway between the International Congresses.

As a step towards filling a need which exists in those areas not catered for by these Societies, at the instigation of Professor Kempo Tsukamoto, President of the International Society of Radiology, the *Asian and Oceanian Society of Radiology* was formed at the conclusion of the Twelfth International Congress of Radiology (Tokyo, 1969).

The sphere of influence of the new Society will extend from the Hawaiian Islands in the East to Pakistan in the West and as far North as Japan and South Korea and as far South as Australia and New Zealand.

At the Inaugural Meeting in Tokyo delegates from 12 countries in the region accepted the invitation of the College of Radiologists of Australasia to conduct the First Congress of the Society in Melbourne, Australia, November 22–26, 1971. Professor W. S. C. Hare, of Melbourne, Australia, was appointed President of the Society and Dr. J. J. Martin, also of Melbourne, Honorary Secretary.

Although this is a Regional Congress, an invitation is extended to Radiologists in other parts of the World to attend.

For further literature concerning the Congress please write to: The Secretary, First Asian and Oceanian Congress of Radiology, Box 805F, G.P.O., Melbourne, 3001, Australia.

SOUTHERN MEDICAL ASSOCIATION RADIOLOGY REFRESHER COURSES

Postgraduate Instruction

The Sixty-fifth Annual Meeting of the Southern Medical Association will be held in Miami Beach, Florida, November 1–4, 1971.

At that time the Radiological Society of North America, in cooperation with the Section on Radiology of the Southern Medical Association, and as a part of a continuing program in postgraduate education, will sponsor four refresher courses.

Two of the courses will be presented by Harley C. Carlson, M.D., Rochester, Minnesota, "The Esophagus." One course will be presented by Arthur R. Clemett, M.D., Miami, Florida, "Radiology of the Liver," and one course by Komanduri Charyulu, M.D., Miami, Florida, "Radiotherapy and the Gastrointestinal Tract."

The instruction sessions will be held at The Fontainebleau Hotel in Miami Beach on Monday, November 1, from 10:30 A.M. to 12 P.M., and from 1:30 P.M. to 5:00 P.M., and on Wednesday, November 3, from 1:30 P.M. to 3:00 P.M.

Attendance is limited to physicians, including graduate students and residents in radiology, and medical students certified by the Dean of their medical school. There will be no registration fee for these courses but all who attend must be registered at the meeting so that they may be properly identified.

Any comments concerning the courses or suggestions for future presentations should be directed to the Co-Chairman of the Refresher Course Committee: Luther W. Brady, Jr., M.D., 230 North Broad Street, Philadelphia, Pennsylvania 19102.

SYMPOSIUM ON NUCLEAR MEDICINE

This symposium under the auspices of

the Central Chapter of the Society of Nuclear Medicine will be held at Indiana University Medical Center, Indianapolis, Indiana, on Wednesday, Thursday, and Friday, September 15–17, 1971.

The curriculum will stress the basic sciences including physical principles, instrumentation, radiobiology, and radiopharmaceuticals, as well as review of the common therapeutic and diagnostic procedures with radionuclides.

Because of the general nature of the course, a limited number of technologists can be registered.

For further information please contact: Office of Post Graduate Medical Education, 1100 West Michigan Street, Indianapolis, Indiana 46202.

SYMPOSIUM ON NUCLEAR MEDICINE

A 3 day Symposium on Nuclear Medicine, sponsored by the Department of Radiology, Columbia-Presbyterian Medical Center, will be held at the Waldorf Astoria Hotel, New York City, September 8–10, 1971.

The Program Chairman is Philip M. Johnson, M.D., Professor of Radiology.

The Guest Faculty Members are: Harold L. Atkins, M.D., Lawrence M. Blau, Ph.D., Monte Blau, Ph.D., M. Donald Blaufox, M.D., Ph.D., N. David Charkes, M.D., Giovanni di Chiro, M.D., Gerald S. Freedman, M.D., Leonard M. Freeman, M.D., Alexander Gottschalk, M.D., John S. Laughlin, Ph.D., Lester M. Levy, M.D., Merle K. Loken, M.D., Ph.D., Leonard Rosenthall, M.S., M.D., Edmundo O. Rothschild, M.D., and Richard P. Spencer, M.D., Ph.D.

The Faculty of the College of Physicians and Surgeons, Columbia University, includes: Henry Aranow, Jr., M.D., Paul J. Cannon, M.D., Carl R. Feind, M.D., Paul N. Goodwin, Ph.D., John H. Laragh, M.D., Raffaele Lattes, M.D., Edith H. Quimby, Sc.D., James A. Reilly, M.D., Richard S. Rivlin, M.D., Edward B. Schlesinger, M.D., Kenneth Sterling, M.D., and Sidney C. Werner, M.D.

The Symposium is designed for physicians engaged in the practice of Nuclear Medicine and will cover as thoroughly as possible most topics of the field. The faculty has been selected to provide authoritative information on the wide range of subjects comprising the program. The Symposium is supported in part by a grant-in-aid from E. R. Squibb & Sons, Inc.

Informal Workshops are held on Thursday afternoon, September 9, in two consecutive groups of 6 simultaneous sessions each.

For further information please write to: Philip M. Johnson, M.D., Columbia-Presbyterian Medical Center, Box No. 153, 622 West 168th Street, New York, New York 10032.

BOOK REVIEWS

The Heart and Its Action: Roentgenky-

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radiology. I doubt, however, that it will stimuthat a qualified teacher conduct the instruction.

The order of presentation of material in *Modern Radiation Physics* is most satisfactory. It begins with an introduction to the basic principles of physics as they apply to radiology. Complete sections explaining therapy, nuclear medicine, diagnosis and protection ensue; and a collection of supplemental tables concludes the text.

Modern Radiation Physics is very readable. It contains an excellent collection of descriptive graphs and tables. Most impressive are illustrative photographs that are generously interspersed so that they spare the reader the task of forming mental images from complicated facts. One can only conclude that this text is well written and useful to all students of radiology.

It must be stressed, however, that it is not a "self-learner." Consequently, someone who is well qualified in teaching radiation physics must direct the use of this text and avail himself of the many questions that will engender it. Only then will the reader experience an improved learning situation.—

Paul M. Kroening M.D.

BOOKS RECEIVED

The Brain of the Oppossum (Didelphis Marsupialis): A Cytoarchitectonic Atlas in Stereotaxic Coordinates. By E. Oswaldo-Cruz and C. E. Rocha-Miranda, with the technical assistance of Raymundo F. Bernardes, Institute de Biofísica, Universidade Federal do Rio de Janeiro, Brasil. Cloth. Pp. 99, with many illustrations. Price, $20.75. Williams & Wilkins Company, Baltimore, Md. 21202, exclusive U. S. agents, 1968.

The Life of Wilhelm Conrad Röntgen: Discoverer of the X Ray. By W. Robert Nitske. Cloth. Pp. 355, with some illustrations. Price, $8.50. University of Arizona Press, P. O. Box 3398, College Station, Tucson, Ariz. 85700, 1971.

Tracer Probes in Steady State Systems. By Robert Steele, Ph.D., Senior Biochemist, Brookhaven National Laboratory; Adjunct Professor of Pharmacology, New York University School of Medicine, New York, N. Y. Cloth. Pp. 236, with some figures. Price, $17.00. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62703, 1971.

Angiologie und Szintigraphie bei Knochen- und Gelenkerkrankungen. Vorträge anlässlich des 50. Deutschen Röntgenkongresses Stuttgart, May 8–11, 1969. Edited by Rolf Glauner. Paper. Pp. 159, with 128 figures. Price, DM 48.–. Georg Thieme Verlag, Stuttgart. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York, N. Y. 10016, 1971.

Leistungsphysiologie: Physiologische Grundlagen der Arbeit und des Sports. By Jürgen Stegemann. Paper. Pp. 249, with 125 figures. Price, DM 9.80. Georg Thieme Verlag, Stuttgart. In the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York, N. Y. 10016, 1971.

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Buffalo Radiological Society

Secretary, Dr. Glen M. Ebersole, 405 Spring St., Jamestown, N.Y. 14701. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiological Society, California Chapter of ACR

Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

Catawba Valley Radiological Society

Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society

Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society

Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society

Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society

Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR

Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society

Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society

Secretary-Treasurer, Dr. Fred H. Dunn, 5940 Forest Park Rd., Suite 101, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR

Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society

Secretary-Treasurer, Dr. John C. Osmer, 20103 Lake Chabot Rd., Castro Valley, Calif. 94546. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society

Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR

Secretary, Dr. Arthur R. Miller, North Miami General Hospital, 1701 N.E. 127th St., North Miami, Fla. 33161. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society

Secretary-Treasurer, Dr. Allen L. Sheer, University Community Hospital, 13505 N. 31st St., Tampa, Fla. 33612. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR

Secretary, Dr. Walker Harris, The Medical Center, Columbus, Ga. 31902. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society

Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society

Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society

Secretary-Treasurer, Dr. George J. Meyer, Holy Cross Hospital, Ft. Lauderdale, Fla., 33308. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists

Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Ghim L. Yeoh, Prof. Center Bldg., 1481 S. King St., Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 P.M.

Health Physics Society

Secretary. John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Waldorf Astoria Hotel, New York City, July 11–15, 1971.

Houston Radiological Society

Secretary, Dr. Milton L. Wagner, 6621 Fannin, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR

Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR

Secretary, Dr. Jack L. Melamed, Grant Hospital of Chicago, 551 Grant Pl., Chicago, Ill. 60614. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR

Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR

Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society

Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society

Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society

Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society

Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society

Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR

Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR

Secretary, Dr. Nathan Stofberg, 4519 Hawksbury Rd., Pikesville, Md. 21208.

Massachusetts Radiological Society, Chapter of ACR

Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101. Biennial Symposium at the Holiday Inn, Bozeman, Mont., July 28–30, 1971.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. N. Patrick Kenney, 3618 S. 114th Ave., Omaha, Nebr. 68144. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89502.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Donald A. Wolfel, Albuquerque, New Mexico. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary-Treasurer, Dr. Samuel H. Madell, 1 E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference: Waldorf Astoria Hotel, New York. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Thomas Lee Taylor, Department of Radiology, University of Florida, Gainesville, Fla. 32601. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Barbara Chick, Glens Falls Hospital, Glens Falls, N.Y. 12801. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiological Society
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Edward I. Miller, 301 Newport Blvd., Newport Beach, Calif. 92660. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Gerald L. Warnock, 11699 N. E. Glisan St., Portland, Ore. 97220. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd.; Beaverton, Oregon 97005. Meets annually in Portland, Oregon, Seattle, Washington or Victoria or Vancouver, British Columbia, in early May.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR

Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary, Dr. Ralph B. Bergerson, 154 Brockenbraugh Ct. Metairie, La. 70005. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07206. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 19–21, 1971.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Lee F. Rogers, Department of Radiotherapy, Bexar County Teaching Hospital, 4502 Medical Drive, San Antonio, Texas. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, 20 Bridge Rd., Kentfield, Calif. 94904. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Raymond L. Schwinn, 480 Monterey Ave., Los Gatos, Calif. 95030. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212. Annual meeting: Miami Beach, Fla., Nov. 1–4, 1971.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Sheraton Hotel, Boston, Mass., September 26–27, 1971.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Los Angeles, Calif., June 26–July 2, 1971.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Haakon O. Haugan, 716 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Sixteenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 28–30, 1972.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Marriott Hotel, Houston, Tex., March 23–25, 1972.

The Fleischner Society
Secretary-Treasurer, Eric N. C. Milne, M.B., Medical Sciences Bldg., University of Toronto, Ontario, Canada.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. R. Newell Ford, St. Mark Hospital, 803 North 2nd West, Salt Lake City, Utah 84103.

Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

VERMONT RADIOLOGICAL SOCIETY, CHAPTER OF ACR

CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.

Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario

[redacted]

Secretary, Dr. Ricardo Cortez, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raúl Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto

Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française de Neuroradiologie
Secretary General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting: Dec. 1971.

Československá Radiologická Společnost

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ABSTRACTS OF RADIOLOGICAL LITERATURE

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ROENTGEN DIAGNOSIS

HEAD

BELL, RUSSEL S., and LOOP, JOHN W. The utility and futility of radiographic skull examination for trauma. *New England J. Med.*, Feb., 1971, *284*, 236–239. (Address: Dr. J. W. Loop, Neuroradiology Section, Department of Radiology, University of Washington School of Medicine, Seattle, Wash. 98105.)

In the face of rapidly rising costs of medical care, systems must be developed to determine whether a diagnostic procedure has been of value in a given case and whether basic medical resources are being utilized efficiently. The authors point out that, although it is ethically displeasing to put a price tag on disease, funds and personnel for health care are not unlimited, and at some point a sufficiently small yield should justify the decision to *not* perform an examination.

At University Hospital and Harborview Medical Center in Seattle a prospective study of skull trauma spanned a 14 month period of time and 1,500 skull roentgenographic examinations. When requesting an examination, house officers were required to fill out a questionnaire which outlined their evaluation of the severity of injury, their predicted odds that a fracture would be found, and their primary reason for requesting the examination. Specific findings in the history, physical examination and neurologic examination had to be indicated. The roentgenograms were then evaluated for skull fracture and for mid-line shift. The questionnaire for head trauma, the roentgenologic analysis, and the follow-up clinical information were then punched on data cards for correlation.

Ninety-three fractures were found in the 1,500 skull examinations. On the basis of preliminary clinical information, patients could be assigned to high-yield groups (92 fractures in 1,065 examinations) or low yield groups (1 fracture in 435 examinations). Furthermore in 65 of the 93 patients with skull fracture, the presence of the fracture did not alter clinical management, which proceeded on the basis of the presence or absence of intracranial injury.

In the 28 patients whose course of therapy was altered because of demonstrable fracture, prophylaclic antibiotics were instituted in basilar skull fractures and surgical operation was performed for depressed fracture fragments. Such altered therapy attempts were to prevent meningitis and epilepsy—complications that develop in a minority of even untreated patients.

Current indications for roentgenographic skull examination in the setting of head trauma are poorly defined. In analyzing the clinical questionnaires, the authors were able to create a list of 21 high-yield findings which could be used as a filter among potential candidates for skull series. Had the presence of at least one of these findings been made a necessary condition for obtaining skull examination, only 1 of the 93 patients with skull fracture would not have had roentgenograms taken (in that patient, therapy was not altered by the roentgenographic documentation); 434 patients without fractures would not have had the skull roentgenograms. If this ratio were applied to skull trauma roentgenograms throughout the entire country, it is estimated that the strategy would result in a savings of $15–20 million a year.—*R. H. Troupin, M.D.*

BLOOMFIELD, J. A. Cloverleaf skull and thanatophoric dwarfism. *Australasian Radiol.*, Nov., 1970, *14*, 429–434. (Address: Senior Radiologist, Royal Hobart Hospital, Hobart, Tasmania.)

The author reports a case of cloverleaf skull deformity associated with thanatophoric dwarfism. The roentgenologic and autopsy findings are recorded and illustrated.

A brief discussion as to the speculative mechanism of the grotesque skull deformity is presented.—*Victor G. Mikity, M.D.*

TAY, C. H. Porencephaly, nasofrontal mucoceles, hypertelorism and segmental vitiligo: report of a new neurocutaneous disorder. *Singapore M. J.*, Dec., 1970, *11*, 253–257. (From: Medical Unit II, Outram Road General Hospital, Singapore 3.)

A neurocutaneous syndrome comprised of porencephaly, nasofrontal mucoceles, ocular hypertelorism and segmental vitiligo is reported.

A case with the above findings and multiple congenital malformations is described which may well be due to ectodermal defect.—*Victor G. Mikity, M.D.*

NECK AND CHEST

McEWEN, J., FINLAYSON, ANGELA, MAIR, A., and GIBSON, A. A. M. Mesothelioma in Scotland. *Brit. M. J.*, Dec., 1970, *4*, 575–578. (From: Department of Social and Occupational Medicine, University of Dundee, Scotland.)

A retrospective study of 80 cases of mesothelioma in Scotland between 1950 and 1967 was carried out to determine the incidence of this disease in Scotland, and the reported relationship to asbestos exposure. The diagnosis was confirmed pathologically in all cases, and no cases in which metastatic pleural or peritoneal disease was suspected were included.

Two sets of control patients were established: (1) the first group represented the baseline of asbestos exposure in the population (patients whose death was unrelated to asbestos exposure-coronary artery disease); (2) the second control group represented a disease (carcinoma), in which there is some suspected relationship to asbestos exposure (lung and gastric carcinoma were matched with pleural and peritoneal mesothelioma, respectively). Each case of mesothelioma was matched as closely as possible with a case from each of the 2 control groups of similar age, date of death, sex, and geographic location. Cases and controls were extensively evaluated and compared for exposure to asbestos, and rated *none*, *possible*, *probable*, and *definite*.

The study shows that the incidence of mesothelioma in Scotland is similar to that in other parts of the United Kingdom. Analysis of asbestos exposure shows a very significant difference between the mesothelioma cases and both sets of controls, when definite exposure is considered alone and when all grades of exposure are considered. The carcinoma controls fall about midway between the mesothelioma cases and the cardiovascular controls. Evidence of asbestosis was found only in 2 of the cases of mesotheliomas.—*Mark D. Reiss, M.D.*

ANDERSON, C. G. Paraquat and the lung. *Australasian Radiol.*, Nov., 1970, *14*, 409–411. (Address: 211 Gloucester Street, Christchurch, New Zealand.)

This is a case report of a 37 year old man who developed moderately advanced bilateral pulmonary fibrosis after having had a previously normal chest roentgenogram.

He had ingested 2 mouthfulls of paraquat (a weed killer) 18 months previously and had been treated with gastric lavage and observed. He had no apparent symptomatic abnormality at that time. Later studies showed impaired pulmonary function.

Paraquat is poisonous with a wide variation in individual response. It has been shown to cause lung changes and can stimulate an exaggerated repair response, particularly in lung tissue.

The author speculates that the lung changes in this patient may have been due to the paraquat ingestion and suggests that exposure to similar toxins might be considered in studying other patients who develop pulmonary fibrosis.—*George L. Sackett, Jr., M.D.*

BOUSHY, SAMUEL F., HELGASON, AUGUST H., and NORTH, LUCEIL B. Occlusion of the bronchial arteries by glass microspheres. *Am. Rev. Resp. Dis.*, Feb., 1971, *103*, 249–263. (From: Pulmonary Function Laboratory and Radiology Department, Veterans Administration Hospital, Houston, Texas; and the Departments of Medicine, Pathology and Radiology, Baylor College of Medicine, Houston, Texas.)

Attempts to occlude the bronchial artery by ligation of the main bronchial arterial trunk are uniformly unsuccessful. Transection of the main stem bronchus temporarily interrupts the bronchial artery circulation; however, after several days regrowth of vessels adjacent to the severed portion, the systemic flow to the lungs is re-established. Occlusion of the lumen of the major bronchial arteries for a considerable length away from the origin should prevent re-anastomosis and re-establishment of the bronchial circulation. The purpose of this study was to determine the effects of such an occlusion on the bronchial circulation.

The bronchial arteries in 26 adult dogs were selectively catheterized and occluded by glass microspheres of 29 μ, 62 μ, or 200 μ in diameter. The selective catheterization was documented by prior test injection of renografin. The occlusion was later confirmed at necropsy. There was no evidence of significant embolization to other organs.

The aorta was filled with a barium mixture either at the time of death or at 6 weeks after the procedure, at which time all the surviving dogs were sacrificed. Roentgenograms to delineate patency of the bronchial arteries were obtained.

In 20 dogs the pulmonary artery and vein were alternately perfused with chrome yellow medium or monastral red at necropsy. The chrome yellow medium fills to the level of the large arterioles, while the monastral red penetrates to the capillary level.

Occlusion of the bronchial arteries by glass microspheres 62 μ in diameter resulted in death of all 5 animals within 48 hours. Six out of the 7 dogs died after embolization with 29 μ microspheres. This second group of dogs generally died at a later time. Two out of 6 dogs died after embolization with 100 μ microspheres, while 1 out of 8 died after embolization with 200 μ microspheres. In dogs dying within 12 days, postmortem aortograms showed that both bronchial arteries were occluded in 7; in 4 one of the bronchial arteries was completely occluded, while minimal flow was noted in the other artery. In 1 dog minimal flow in both bronchial arteries was demonstrated. This latter dog lived 9 days and may have re-established some of the circulation.

In dogs sacrificed at 6 weeks, 2 dogs showed evidence of complete obstruction of one major bronchial artery. These dogs were injected with the 200 μ microspheres. None of the surviving dogs injected with smaller sized particles had complete occlusion of the bronchial arteries. Histologically, the lungs showed inflammation and then later necrosis of the bronchus and surrounding lung. Monastral red was noted in the peripheral bronchial vessels when it was infused in either the pulmonary artery or the pulmonary vein; however, there was no evidence of chrome yellow in the peripheral bronchial arteries.

From the above results the authors conclude that 200 μ microspheres occluded the large bronchial arteries and preserved the smaller arterioles. Glass microspheres of 62 μ were uniformly lethal and indicate that there is a critical size in the bronchial artery system for viable bronchi. The collateral circulation appears to come from both the pulmonary artery and vein via channels that appear to occur at the capillary level rather than the arteriolar level.—*Frederick P. Stitik, M.D.*

Seaton, Anthony, Lapp, N. Leroy, and Chang, C. H. Joseph. Lung perfusion scanning in coal workers pneumoconiosis. *Am. Rev. Resp. Dis.*, March, 1971, *103*, 338–349. (From: Appalachian Laboratory for Occupational Respiratory Diseases, Department of Health, Education, and Welfare, U. S. Public Health Service, Bureau of Occupational Safety and Health; and the Departments of Medicine and Radiology, West Virginia University Medical Center, Morgantown, W. Va.)

In coal workers pneumoconiosis, the primary lesion is an accumulation of coal dust around the respiratory bronchioles which leads to fibrosis. Dilatation of the respiratory bronchioles may result in focal emphysema. In simple pneumoconiosis (mild involvement without conglomerate masses) vascular obliteration is rare unless there has been a secondary infection. In complicated pneumoconioses, however, there may be marked obliteration of the pulmonary arteries and arterioles by direct encasement by the conglomerate masses, or by an endarteritis which is also frequently associated with these mass lesions. Pathologically, conglomerate masses have been shown to be surrounded by plexuses of wide-diameter capillaries (50 μ to 150 μ in diameter). Areas of bullous cysts and compensatory emphysema frequently are seen concomitantly with conglomerate masses.

Physiologic data have suggested that there may be obliteration of small vessels even in simple pneumoconiosis; however, pathologic examination of the lungs of patients with simple pneumoconiosis has failed to demonstrate small artery disease. The present study was undertaken to define the role of small vessel disease in simple pneumoconiosis, and complicated pneumoconiosis.

Thirty-seven volunteer coal miners were studied. Standard chest roentgenograms (72 inch distance, posteroanterior and lateral exposure) were obtained. Two were normal, 14 had large fibrotic masses, and the remaining 21 had simple pneumoconiosis by the UICC.Cincinnati Classification of the Radiographic Appearances of Pneumoconioses. Rectilinear photoscans were obtained in anteroposterior and both lateral projections after the intravenous injection of 300 μc of macroaggregated albumin. The abnormalities were recorded without knowledge of the chest roentgenogram and included: (1) area of decreased activity surrounded by areas of increased activity; (2) localized peripheral indentations of decreased activity (crescents); (3) fissure sign; and (4) absence of normal decrease in activity from the lower to the upper zones.

Pulmonary function tests included spirometry as well as measurements of blood gases, physiologic dead space, venous admixture and diffusing capacity. Thirteen patients underwent right heart catheterization. Detailed histories with regard to occupation, cigarette smoking, and cardiac disease were obtained.

In all 14 patients with conglomerate masses on the chest roentgenogram, the scans revealed a perfusion defect. This included a lesion 1.1 cm. in diameter. Bullae were detected in 6 subjects, while in 4 others areas of underperfusion appeared in areas which were normal on the chest roentgenogram. These probably represented areas of "compensatory" emphysema.

Two patients with Category 3 simple pneumoconiosis had abnormal scans. One showed peripheral crescents of decreased perfusion probably related to silicosis and the other demonstrated a decreased perfusion in an area of heavy nodular infiltration. Of the 19 patients with Category 1 and 2 pneumoconiosis, 12 had normal scans. Four of these patients had peripheral defects corresponding to old tuberculosis or a possible pulmonary embolus, while the remaining 2 had reversal of flow with increased flow to the upper lobes and a decrease in perfusion of the lower lobes. Of the 2 patients with reversal of the flow, 1 was undoubtedly on the basis of pulmonary hypertension documented at cardiac catheterization; the other had no apparent cause, although this may be due to the fact that the patients were injected in an upright position.

Two of the volunteers did not have pneumoconiosis. One had severe chronic bronchitis; his scan showed a bilateral fissure sign with evenly distributed perfusion.

From these data the authors conclude the following:

1. Almost all of the perfusion defects found in patients with simple pneumoconiosis could be explained on the basis of other lesions seen on the chest roentgenogram. Filling defects were seen in one area of very early conglomeration and in another with heavy nodular infiltration, suggesting that vascular obstruction may occur early in progressing pneumoconiosis.

2. In complicated pneumoconiosis, nodules seen on the chest roentgenogram corresponded in size and configuration to perfusion defects on the scans. All but 1 of the patients had physiologic evidence of an arteriovenous shunt but this did not correspond in size and probably was not the cause of the perfusion defect. The absence of radioactivity in the liver and spleen also indicated that significant pulmonary to systemic shunting around the conglomerate masses was not present.

3. Bullae and compensatory emphysema may appear on scans as areas of perfusion defect in regions of the lung which appear normal on the chest roentgenogram. The scan may be a more sensitive test to detect these early abnormalities.—*Frederick P. Stitik, M.D.*

Yano, Y., McRae, J., and Anger, H. O. Lung function studies using short-lived ^{81m}Kr and the scintillation camera. *J. Nuclear Med.*, Nov., 1970, *11*, 674–679. (From: Donner Laboratory of Medical Physics and Biophysics, University of California, Berkeley, Calif.)

For further development of generator produced short Kr^{81m} lived radioisotopes, the authors have investigated the radioactive gas Kr^{81m}. A method is presented for obtaining millicurie amounts of Rb^{81} for both pulmonary perfusion and the ventilation studies.

To evaluate the physiologic and clinical informa-

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cineroentgenography, and is delineated by animation techniques.

During the course of the meal, 10 barium spheres are swallowed. They pass quickly into the stomach. More food is then taken, after which 10 rods are swallowed. They do not always follow the lesser curvature, the so-called "Magenstrasse" of the stomach.

Foods ingested during the course of the meal show various arrangements in the stomach depending on the gastric shape, the pressure of gas in the colon and the time factor. Under fluoroscopy, it is noted that swallowed foods are not mixed in the stomach immediately, but show 'layer formation,' although incomplete.

Fifteen minutes after ingestion, food is arranged in the same layers as it was at the end of the meal.

After *30 minutes*, the swallowed foods are moved *in toto* slightly toward the antrum. Changes in body position or pressure do not contribute significantly to mixing of the stomach contents.

series. Diverticulum was demonstrated in 5 patients, hamartoma of the small bowel was identified in 2 patients, ileal polyp and hyperplasia of lymphoid tissue were found in the others.

Abdominal pain was the initial symptom in 75 per cent of the patients, vomiting occurred in 70 per cent and bleeding per rectum or blood and mucus in the stools was reported in 52 per cent. Constipation was present in 72 per cent and diarrhea in 11 per cent. An abdominal mass was felt in 68 per cent of the patients and a mass was felt in the rectum in 6 per cent. Abdominal pain, vomiting, bleeding per rectum and/or blood on the examining finger and a palpable mass made the diagnosis obvious in more than two-thirds of the patients. The picture was not so typical in the remaining patients. Colic was not always an outstanding feature and blood was not passed per rectum until several hours after the onset.

The abdomen usually showed absence of gas from the right side of the abdomen. Occasionally the outline of an intussusception may be seen on the roentgenogram. Multiple fluid levels may be observed in late cases. The barium enema will outline the apex of intussusception as a filling defect within the contrast enema. Fifteen patients had undergone spontaneous reduction at laparotomy.

Simple operative reduction was carried out on 140 patients with 2 deaths—a mortality rate of 1.42 per cent. Operative reduction and appendectomy were carried out in 90 patients with 1 death—a mortality rate of 1.1 per cent. Resection and primary end-to-end anastomosis was carried out in 23 patients for irreducibility, gangrene, or primary tumor of the bowel. There were 4 deaths in this group; *i.e.*, a 17.39 per cent mortality rate. The over-all mortality in the present series was 3.4 per cent, or 10 deaths out of 288 patients.

The relation of the duration of symptoms to irreducibility and mortality is discussed. The group in which the intussusception had been present for more than 49 hours showed an increase in irreducibility and 1 death following resection and primary anastomosis. In the group with duration of symptoms of over 73 hours there were 7 cases irreducible and the mortality rate at surgery was 13 per cent. There was recurrent intussusception present in 14 cases.

The authors also discuss the choice in the treatment of intussusception between operation and hydrostatic reduction using the barium enema. The choice between the 2 methods usually depends upon the general condition of the patient, the duration of the disease and the availability of a 24 hour skilled radiologic service. The results of operative and nonoperative reduction are equally good as there is now practically no mortality for reducible intussusception. It is concluded that the only valid contraindication to barium enema is the severity, and not the duration, of the child's illness.

The authors report the mortality for resection in this series to be high, 17.3 per cent. Resection and primary anastomosis is the best method of treatment of irreducible intussusception, provided that the surgeon has extensive experience in bowel surgery in children.—*James F. Martin, M.D.*

Massoud, G. E., Farag, A., Hilmy, A., El-Sallab, R., and Ghanem, M. Angiography of the bilharzial liver. *Bull. Alex. Fac. Med.*, June, 1968, *4*, 188–197. (From: Departments of Radiology and Surgery, Alexandria University, Egypt, U. A. R.)

Pathologically the most conspicuous finding in bilharzial liver is the thickening, cellularity, and fibrosis of the portal tracts, with, however, the fundamental lesion appearing to be the occlusive changes in the fourth order of branches of the portal vein. This results in a new vascular network systematically along the portal tributaries, the nature of which has not been described in any other hepatic abnormality. The occlusive changes in the smaller branches of the portal vein, and the periductal plexus of the new vessels are visible on a splenoportogram, which also reveals that there is no narrowing or distortion of the main portal tributaries.

In Laennec's cirrhosis the splenoportogram shows a gross reduction in the size and extent of the portal tree with displacement of its branches by zones of regenerating liver nodules. The newly formed vessels are never regularly dispersed around the main ramifications and do not form a sheath around them as occurs in bilharziasis.

The livers were studied in hospitalized patients by performing splenoportography and retrograde wedge venography through a catheter introduced into the medial cubital vein. The postmortem livers were studied by portal venograms and by casts of the hepatic veins. The postmortem studies confirm that the diminution of the portal venous bed is manifested mainly as an absence of the small fourth degree branching giving the picture of a so-called "Withered Tree." These studies demonstrated also that roentgenographically the most important finding is the presence of an extensive network of small closely interlaced vessels which symmetrically surround the main portal tributaries simulating a mossy proliferation around the trunk and branches of a tree.

It is reportedly a common belief that the systemic hepatic venous system does not share in the process of bilharziasis of the liver, but the systemic venohepatograms in the present study did show marked tortuosity and a diminished caliber of these veins, together with some disturbance of the anatomic pattern.

It is suggested that these observations need further investigation and confirmation.—*Donald M. Monson, M.D.*

Deimer, E. Die perkutane Hepatographie. (The percutaneous hepatography.) *Fortschr. a. d.*

Geb. d. Röntgenstrahlen u. d. Nuklearmedizin, Jan., 1971, *114*, 84–102. (Address: Röntgendiagnostisches Institut der Universität, A-1090 Wien/Österreich.)

An intraparenchymal deposit of contrast medium in the liver is removed rapidly by the vascular structures. By this method it is possible to visualize the vessels in the liver and to obtain information regarding the architecture and dynamics of the vasculature. In addition, it is possible to demonstrate abnormal lympho-dynamics and obstruction to bile flow. Lymphatic visualization takes place in cases with obstruction of the portal or hepatic venous system. An obstructed bile duct causes long lasting opacification of the proximally dilated ducts. Normal ducts are only filled by chance and empty immediately.

A kinetic intrahepatic injection of contrast material under general anesthesia was used. A spinal needle was introduced from the right side between the anterior and posterior axillary line into the superior anterior portion of the liver. This site of injection was chosen to be as far away from the lower surface as possible to avoid puncture of the portal vein or larger bile ducts. During withdrawal of the needle a water soluble contrast medium was injected over a period of 7 seconds. By this method the contrast material is deposited over a large area resulting in opacification of several vessels.

The different hepatic structures such as the portal venous system, the hepatic veins, the hepatic arteries, the bile ducts, and lymphatic vessels can be distinguished by their localization, shape, and caliber as well as by the direction and speed of contrast flow. Also avascular lesions can be detected as they appear as filling defects in the hepatogram phase. Filling of the lymphatic vessels, which are seen as tortuous, beaded structures in the hepatic parenchyma and in the gastroduodenal ligament indicates cirrhosis. However, there was no correlation between lymphatic visualization and duration or stage of the cirrhosis.

The value of this method was illustrated in a normal case and in several cases with characteristic pathologic findings. It is considered a method of small risk, which makes it suitable for routine use, adding a new dimension to the evaluation of liver morphology.—*Peter F. Winter, M.D.*

Dijken, B. G., Hart, H. Ch., Imhof, J. W., and Sluiter, J. T. Benign haemangioma of the liver: the significance of selective angiography. *Radiol. clin. et biol.*, 1971, *40*, 50–55. (From: De Lichtenberg Hospital, Amersfoort, The Netherlands.)

This is a single case report discussing the angiographic appearance of a hemangioma of the liver.

The case illustrates the typical appearance which is well known.—*George L. Sackett, Jr., M.D.*

Glenn, Frank. Obstruction of the common bile duct. *Surg., Gynec. & Obst.*, Jan., 1971, *132*, 25–35. (From: The New York Hospital and Cornell Medical Center, New York, N. Y.)

The normal anatomy and physiology of the biliary tract are described. The pathologic physiology of biliary pain and jaundice is then considered, followed by a brief statement of general principles in biliary surgery.

The clinical manifestations and surgical management of biliary calculi, stricture, and carcinoma are discussed in greater detail, including some case presentations.

There is also a review of congenital anomalies, Caroli's disease (diffuse intrahepatic ductal dilatation), Echinococcus cysts, and iatrogenic injury.—*Mark D. Reiss, M.D.*

Aagenaes, Øystein, Sigstad, Helge, and Bjørn-Hansen, Ragnar. Lymphoedema in hereditary recurrent cholestasis from birth. *Arch. Dis. Childhood*, Oct., 1970, *45*, 690–695. (Address: Dr. Ø. Aagenaes, Department of Paediatrics, University Hospital, Rikshospitalet, Oslo, Norway.)

The authors report an inherited disorder characterized by a combination of lymphedema, and intrahepatic cholestasis in a Norwegian family. The cholestasis was always recognized shortly after birth. The jaundice persisted from 1 year to more than 7 years. During the cholestatic period, all patients had hepatomegaly, abnormal liver function, and a severe malabsorption picture.

Liver biopsy during infancy showed giant cell transformation and intrahepatic cholestasis with slight increase of connective tissue in the periportal tracts and around the central veins. The adult cases showed an intracanalicular cholestasis during the jaundiced periods and a variable degree of increase in connective tissue. Signs of portal hypertension were absent. Some children had slight pedal edema, in the preschool years, and all had somewhat thick, stout legs at this age with slowly progressive edema throughout the school years. The edema may be relieved by elevation of the legs periodically or by diuretics. Recurrent episodes of cholestasis have not affected the tendency to edema; in fact, some patients have thought that the edema improved in the cholestatic phases. Some have had a tendency to lymphangitis.

The lymph vessels in the lower extremities have been investigated in 3 adults and 3 children, including complete lymphangiography of one leg in the adults, and the oldest child. The Patent Blue injected for visualization of the lymphatics tended to show an abnormal intradermal spread. The lymphangiograms were abnormal in all cases examined, with abnormal filling of numerous collateral pathways; dermal backflow delayed clearing of the contrast at 24 hours. One 8 year old girl showed a reduced number of lymphatics in the calf and thigh, but otherwise the study was normal. No abnormality of the thoracic duct or the intraabdominal lymphatics was demonstrated.

The evidence suggests that the lymph vessel pathology is congenital in origin and is the cause of the edema.

No chromosomal abnormalities have been found.

was less than 9, the collecting systems were not visualized. However, when the ratio of urine to plasma creatinine reached a level of 16 or greater, adequate visualization of the collecting systems was achieved.

This was usually seen best in the early roentgenograms and visualization was sometimes evident in 1 minute. Asynchronous filling was common and the fine structure of the calyceal systems was not always visualized, although the pelves, ureters, and

[redacted]

have not been encountered. Hemorrhage or hematoma formation related to biopsy or surgery may be

[redacted]

Robert M. Acute renal failure following intravenous urography in patients with

[redacted]

thrombosis, urinary obstruction or inflammatory process. Changes in the vicinity of the transplanted kidney such as displacement and dilatation of the arteria circumflexa ilei profunda or a blush of granulation tissue in the new bed of the transplant, are considered as reliable findings of rejection.

Complications due to immuno-suppressive therapy are discussed insofar as they are significant in roentgenologic diagnosis.—*Peter F. Winter, M.D.*

DORNFELD, LESLIE, LECKY, JAMES W., and PETER, JAMES B. Polyarteritis and intrarenal renal artery aneurysms. *J.A.M.A.*, March 22, 1971, *215*, 1950–1952. (Address: Dr. J. B. Peter, Department of Medicine, UCLA School of Medicine, Los Angeles, Calif. 90024.)

The diagnosis of polyarteritis nodosa may be difficult to establish in the work-up of a patient with a multi-system disorder. Polyarteritis involves the kidney in 80 per cent of autopsy-proved cases and for this reason renal biopsy has been one of the diagnostic procedures commonly performed. Because of the associated hypertension and azotemia in these patients, however, the risk of renal biopsy is increased, and an alternative method of establishing the diagnosis would be extremely valuable.

Renal arteriography has been previously reported to show multiple small eccentric aneurysms of the distal segmental, interlobar, and arcuate arteries. The pathogenesis of these is related to fibrinoid necrosis which weakens layers of the arterial wall during the acute phase. Other changes which may occur during the course of the disease include intraparenchymal and subcapsular renal hemorrhage, as well as thrombosis leading to ischemia, infarction and scarring.

The authors report 3 additional cases studied at UCLA to illustrate these features. In their previous experience with renal arteriography (300 cases) the only other patient showing similar intrarenal aneurysms had fibromuscular dysplasia of the main renal artery. They feel, therefore, that in the absence of large vessel disease, the arteriographic findings are specific for polyarteritis.—*R. H. Troupin, M.D.*

BLOOD AND LYMPH SYSTEM

FALLAT, ROBERT J., POWELL, MALCOLM R., YOUKER, JAMES E., and NADEL, JAY A. Pulmonary deposition and clearance of ^{131}I-labeled oil after lymphography in man: correlation with lung function. *Radiology*, Dec., 1970, *97*, 511–520. (From: Cardiovascular Research Institute, Department of Radiology, and the Nuclear Medicine Section, University of California San Francisco Medical Center, San Francisco, Calif.)

Lymphography is a well-established radiographic diagnostic procedure for investigating the spread of certain malignancies. Further, the therapeutic injection of radioiodinated oil into the lymphatic channels has been proposed.

The secondary pulmonary embolism by the oily agents, and the possible complications attendant to this, form the basis for presentation. Five patients who had intralymphatic injections of ethiodol labeled with I^{131} were studied.

The authors believe that they can: (a) characterize the distribution and clearance of oily emboli in the lung; (b) relate the changes in pulmonary function to the quantity of oil in the lung; and (c) estimate the radiation hazard to the lung from isotope-labeled oil.

The distribution patterns of accumulated radioactivity within the lungs of all 5 patients were obtained by the rectilinear scanner or scintillation camera examinations. These were begun within 3 to 24 hours of the injection of the radioiodinated oil, and repeated every 1 to 2 days for the first week, and at longer intervals thereafter for as long as 19 days. Because of the obvious similarities between the oil embolism and pulmonary perfusion study with I^{131} MAA, the latter was done on 2 of the 5 patients after their lungs had cleared the ethiodol I^{131}.

Assuming the total counts of I^{131} MAA in the lungs proportional to the total quantity injected, the amount of ethiodol I^{131} (in millicuries) was calculated by the formula:

$$\frac{\text{mc I}^{131}\text{ MAA injected}}{\text{counts I}^{131}\text{ MAA}}.$$

An estimation of the total body retention was done serially in 3 of the 5 patients by means of a 3 inch crystal detector with a straight bore collimator positioned 1 meter from midbody of the standing patient. Prior to lymphography, and on the same serial schedule as the scanner and camera examinations, vital capacity, timed vital capacity, and maximal flow rates were determined spirometrically. By plethysmography, total lung capacity and airway resistance were also measured. Simultaneously, single-breath carbon monoxide diffusing capacity and neon-dilution total-lung capacity were ascertained.

The accumulated information revealed that following lymphography with ethiodol I^{131} radioactivity could be detected in the lungs of all 5 patients. This was maximal within 24 hours, and 40 per cent or more of the injected dose was present in the lungs of 3 patients. The difference of the embolized oil mimicked that of I^{131} MAA, but there was one difference. The upper lung fields contained a smaller fraction of the embolized oil than I^{131} MAA for the first 5 days postinjection. Thereafter this seemed to change, and the upper lung fields cleared the oil less rapidly than did the rest of the lung. Actually, clearance of the radioiodinated oil was slow in all 5 patients, and varied from one to another. The half-life of I^{131}, in the lungs, was determined after it had reached a maximum on Day 1, and varied from 5.2 to 12.6 days. There was definite impairment in pulmonary function in all patients but it was not marked. This was primarily reflected as a decrease in CO diffusing capacity in all patients. Further, this was biphasic in 1 patient occurring first when the concentration of oil in the lungs was maximal and again on the 19th day when only a small percentage (16.5 per cent) of the oil remained in the lungs. This was not so obvious in the other 4 patients, but variations did occur which indicate that there may be some other mechanism besides the physical presence of the oil to explain the decrease in diffusing capacity. Using the minimum parameters of 50 per cent of the injected oil embolizing to the lungs and an effective half-life of 3 days, the authors have calculated a radiation dose to the lung of 34 rads/mc of I^{131} injected into the lymphatics. They also point out that the capillary endothelium, which is the most radiosensitive tissue in the lung, would receive the greatest exposure.

This study has revealed some very significant facts which should be of interest to those performing lymphography. Some of the causative mechanisms, especially relating to decreased diffusing capacity, must yet be determined.—*F. C. Petty, M.D.*

RADIATION THERAPY

Furnival, Isobel G., Stewart, Helen J., Weddell J. M., Dovey, P., Gravelle, I. H., Evans, K. T., and Forrest, A. P. M. Accuracy of screening methods for the diagnosis of breast disease. *Brit. M. J.*, Nov., 1970, *4*, 461–463. (Address: Isobel G. Furnival, M. B., Clinical Research Assistant, Welsh National School of Medicine, Cardiff, Wales.)

As mass screening techniques for the early diagnosis of carcinoma of the breast, clinical examination, thermography and 70 mm. mammography were performed in 891 patients—414 symptomatic and 477 asymptomatic.

The results of this survey indicated that the diagnostic accuracy of thermography and 70 mm. mammography does not approach that of the clinical examination in patients with symptomatic breast disease. While, one clinician correctly diagnosed malignant disease in 82 per cent of women with breast complaints, the best possible accuracy, as represented by the highest individual accuracy when 3 radiologists independently surveyed the films, was only 53 per cent for thermography and 61 per cent for 70 mm. mammography.

It is considered a dangerous policy to use thermography or 70 mm. mammography as isolated screening procedures for breast cancer.—*A. Franklin Turner, M.D.*

Jelliffe, A. M., Millett, Yvonne L., Marston, J. A. P., Bennett, M. H., Farrer-Brown, G., Kendall, B., and Kelling, D.H. Laparotomy and splenectomy as routine investigations in the staging of Hodgkin's disease before treatment. *Clin. Radiol.*, Oct., 1970, *21*, 439–445. (From: Departments of Radiotherapy, Surgery, Pathology, Radiodiagnosis and Nuclear Medicine, Middlesex Hospital, London, W.1, and Mount Vernon Hospital, Northwood, Middlesex, England. Based on material reported by the Working Party of the Clinical Cancertherapy Cooperative Group, February, 1970.)

Twenty-two patients with Hodgkin's disease in whom laparotomy, splenectomy, abdominal lymph node biopsy and open liver biopsy have been performed during routine investigation are reported. The extent of the disease so revealed was compared with the extent of the disease thought to be present before laparotomy as determined by clinical, radiologic, isotopic and biochemical investigations.

It can be concluded that the lymphangiogram is a valuable but not infallible diagnostic method. Grossly abnormal lymph nodes were always confirmed at laparotomy. Of 11 negative lymphangiograms only 1 proved to be incorrectly diagnosed. In 12 patients there was no correlation of splenic involvement preoperatively and postoperatively. It would seem that it is impossible to make a definite diagnosis of either a normal or abnormal spleen without performing a splenectomy. Liver involvement is impossible to diagnose otherwise than by direct study.

On the basis of this detailed investigation the authors feel that in the near future laparotomy, splenectomy, abdominal lymph node biopsy and liver biopsy will be generally accepted as essential routine procedures before attempting to cure patients with Hodgkin's disease using radical radiotherapy.—*S. A. Kaufman, M.D.*

Kyle, Robert A., Pierre, Robert V., and Bayrd, Edwin D. Multiple myeloma and acute myelomonocytic leukemia: report of four cases possibly related to melphalan. *New England J. Med.*, Nov., 1970, *283*, 1121–1125. (From: Division of Internal Medicine and Hematology and Department of Clinical Pathology, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

This is a report of 4 cases who received melphalan for a prolonged time and developed acute myelomonocytic leukemia. Myelomonocytic leukemia is used here as a designation for acute leukemia that involves more than one marrow cell line.

Three patients were being treated for typical multiple myeloma (30, 33 and 44 months) and the fourth for systemic amyloidosis (5 years).

[redacted]

fluid and cardiac position. *J. Nuclear Med.*, Jan., 1971, *12*, 2–4. (From: St. Mary's Hospital and Claire Zellerbach Saroni Tumor Institute, Mt. Zion Hospital and Medical Center, San Francisco, Calif.)

With the dual probe rectilinear scanner, one lung is scanned in the dependent position, while the other is scanned in the nondependent position.

The appearance of the lungs in patients with free pleural fluid and/or an enlarged heart using the lower probe on the side with effusion is compared to that using the upper detector for the scan. The changes that occur in the lateral decubitus roentgenograms document the shift in the position of the apex of the heart from a 3 cm. to a 1 cm. separation from the left lateral chest wall in the right and left lateral decubitus position, respectively.

The cardiac impression was greatly accentuated secondary to displacement of a left lower lobe activity in the dependent lateral decubitus scan.

The pattern obtained with pleural fluid in the dependent position was one of diminished activity in the lower lobes and along the fissures, and correlated well with the roentgenographic appearance of free fluid. In the dependent position, the fluid insinuated itself between the lobes and separated them. The separation along the major and minor fissures could be clearly delineated.

When scanning patients for a clinically suspected embolism, it is important not to confuse the patterns of free fluids with those of perfusion defects. The differentiation between free fluid and embolism can be made by rescanning the lung in question in the nondependent position.—*H. William Strauss, Major, USAF, MC*

ZOOK, ELVIN G., BOLIVAR, JUAN C., and EPSTEIN, LEONARD I. The value of scintiscans in the diagnosis of splenic abscess. *Surg., Gynec. & Obst.*, Dec., 1970, *131*, 1125–1129. (From: Department of Surgery, Indiana University Medical Center and the West Tenth Street Veterans Administration Hospital, Indianapolis, Ind.)

Since splenic abscesses are infrequent, the possibility is often not considered in the differential diagnosis of left sided abdominal or low chest pain combined with evidence of infection.

Three cases are reported.

In 2 patients splenic scanning had been used as one of the methods of diagnosis, and showed an area of decreased uptake at the sites of the abscesses. These 2 patients were explored; splenectomy was performed and the patients recovered.

In a third case, in whom scanning had not been done, and the diagnosis was not established before surgery, the abscess ruptured at the time of exploratory laparotomy; generalized peritonitis and death followed.—*Lois Cowan Collins, M.D.*

SIGEL, RICHARD M., BECKER, DAVID V., and HURLEY, JAMES R. Evaluation of spleen size during routine liver imaging with ^{99m}Tc and the scintillation camera. *J. Nuclear Med.*, Nov., 1970, *11*, 689–692. (From: The New York Hospital–Cornell Medical Center, New York, N. Y.)

A screening test to evaluate the spleen size was derived from the vertical height of the spleen as seen in the posterior view of technetium 99m sulfur colloid liver and spleen scintillation camera image.

The reproducibility of the spleen images was evaluated using: (1) a grid source of various distances from the face of the detector; (2) a spleen phantom; (3) field uniformity determination; and (4) various intensity settings. The maximum error was a 1.0 cm. overestimation in splenic height.

The consecutive liver and spleen images in 316 patients were studied for spleen size. The estimated height of the spleen ranged from 6 to 26 cm., with a mode of 11 cm., and a mean of 11.9 cm.

There were 24 patients who had no evidence of hepatic or splenic disease. The spleen height in these patients ranged from 8 to 14 cm. with a mean of 10.6, plus or minus 1.7 cm. Spleen height alone was selected as an index of spleen size because it is the simplest possible measurement.

A good correlation between spleen height on abdominal roentgenograms and spleen weight had been previously described.

A spleen height of 6 to 14 cm. is considered normal that of 15 to 18 cm. as increased and that of 19 cm or greater as markedly increased, corresponding to a normal, enlarged and markedly enlarged spleen size, respectively, as categorized on the basis of scintillation camera images.—*H. William Strauss, Major, USAF, MC*

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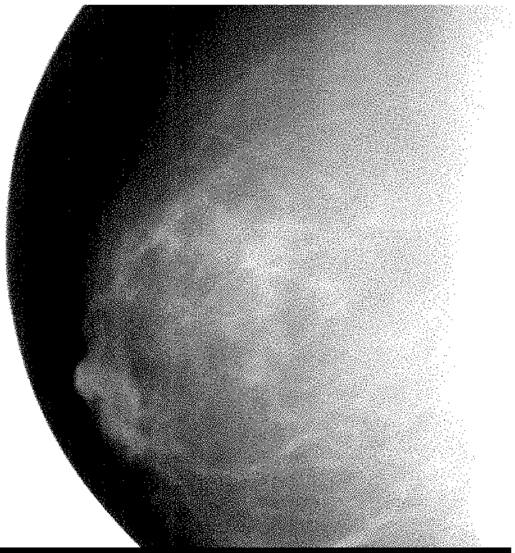

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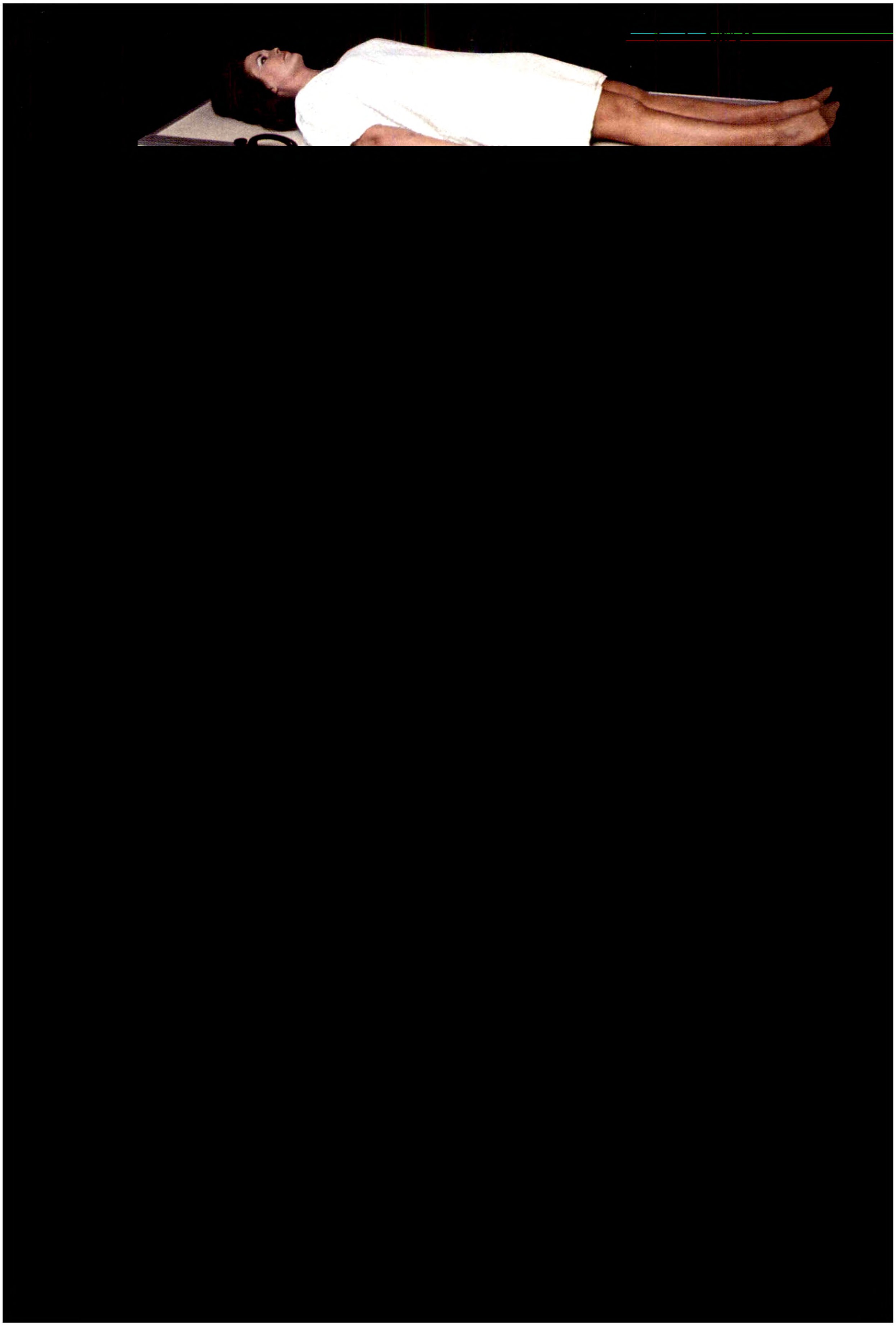

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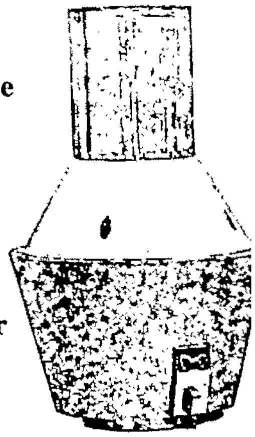

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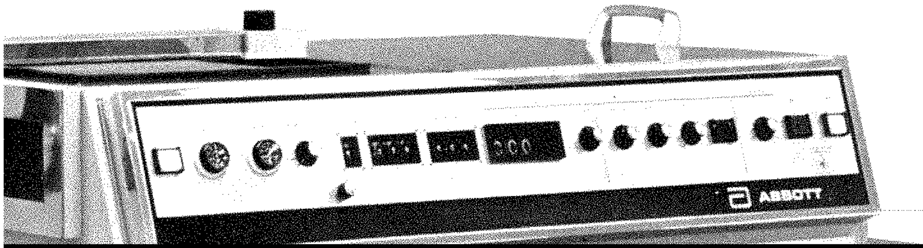

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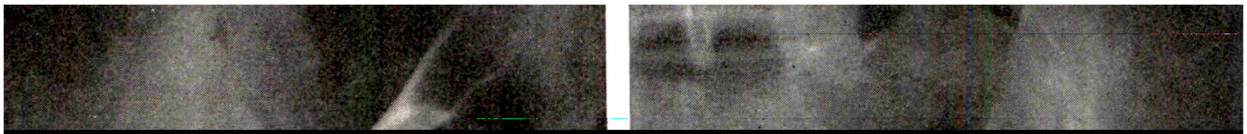

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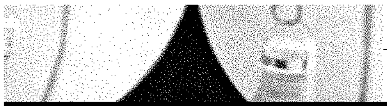

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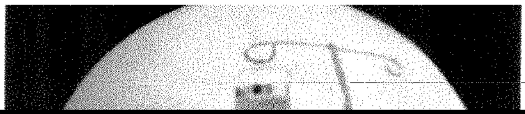

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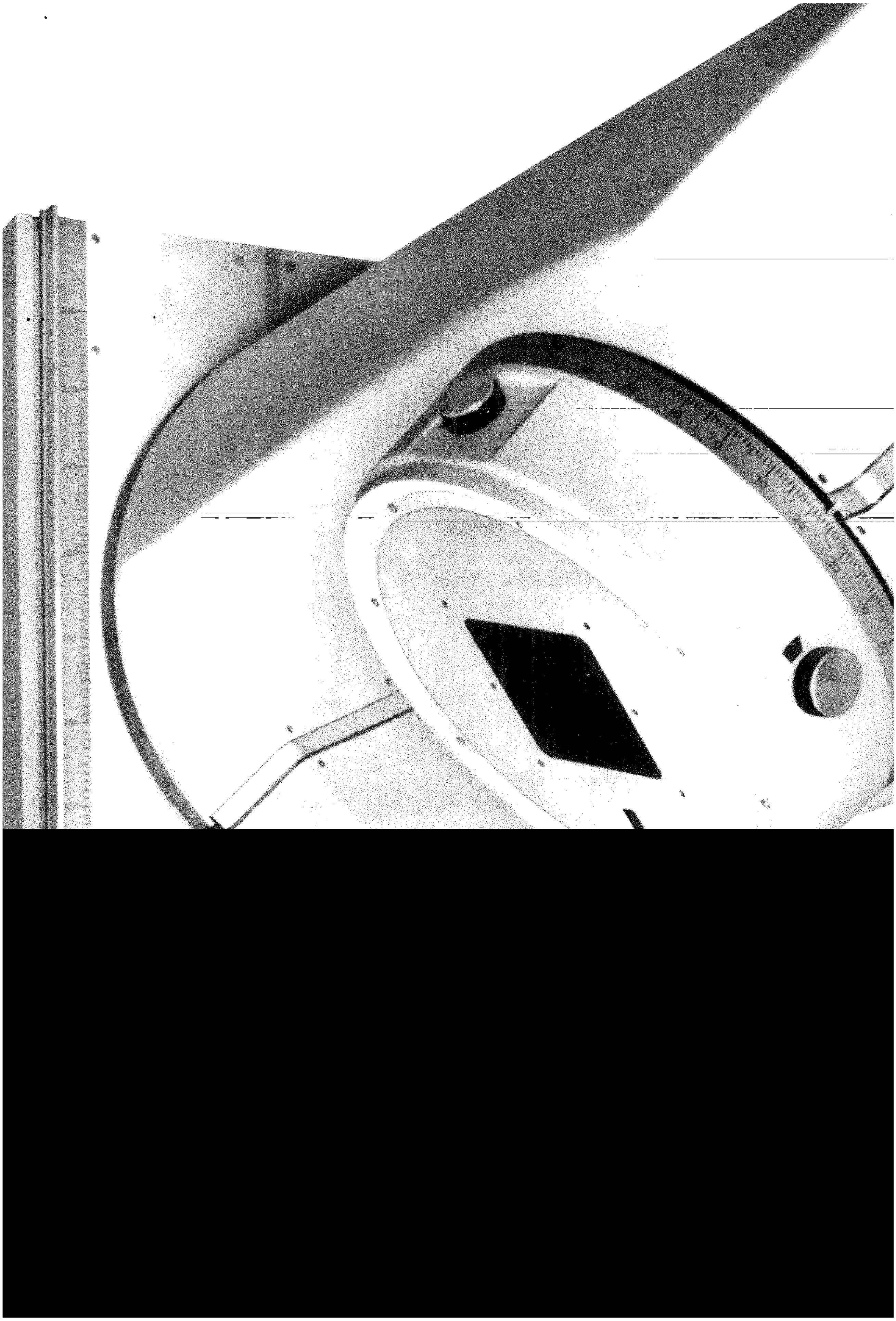

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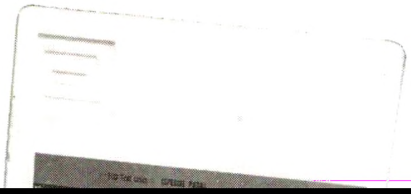

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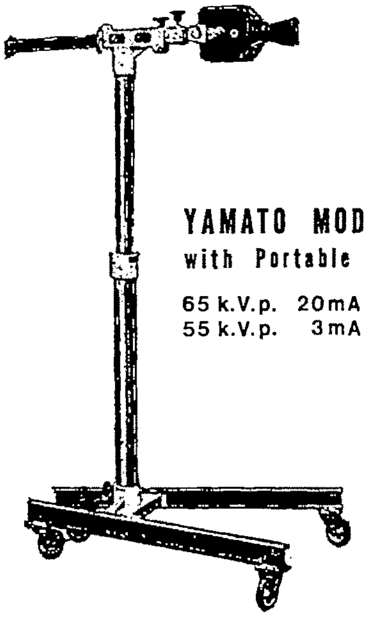

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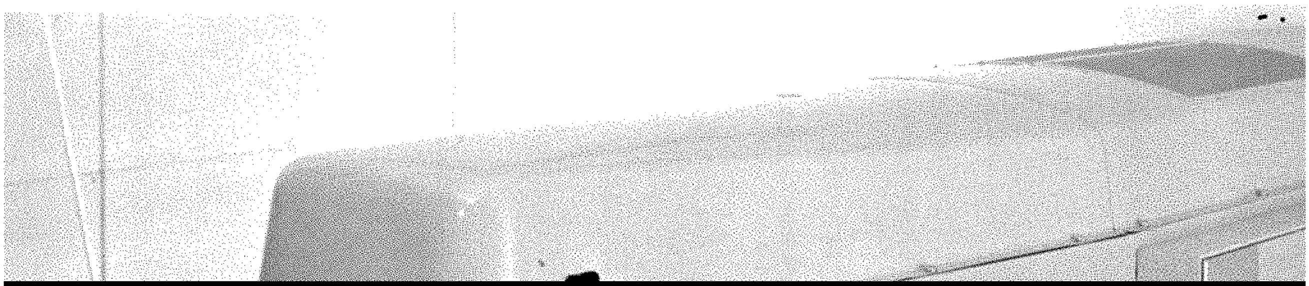

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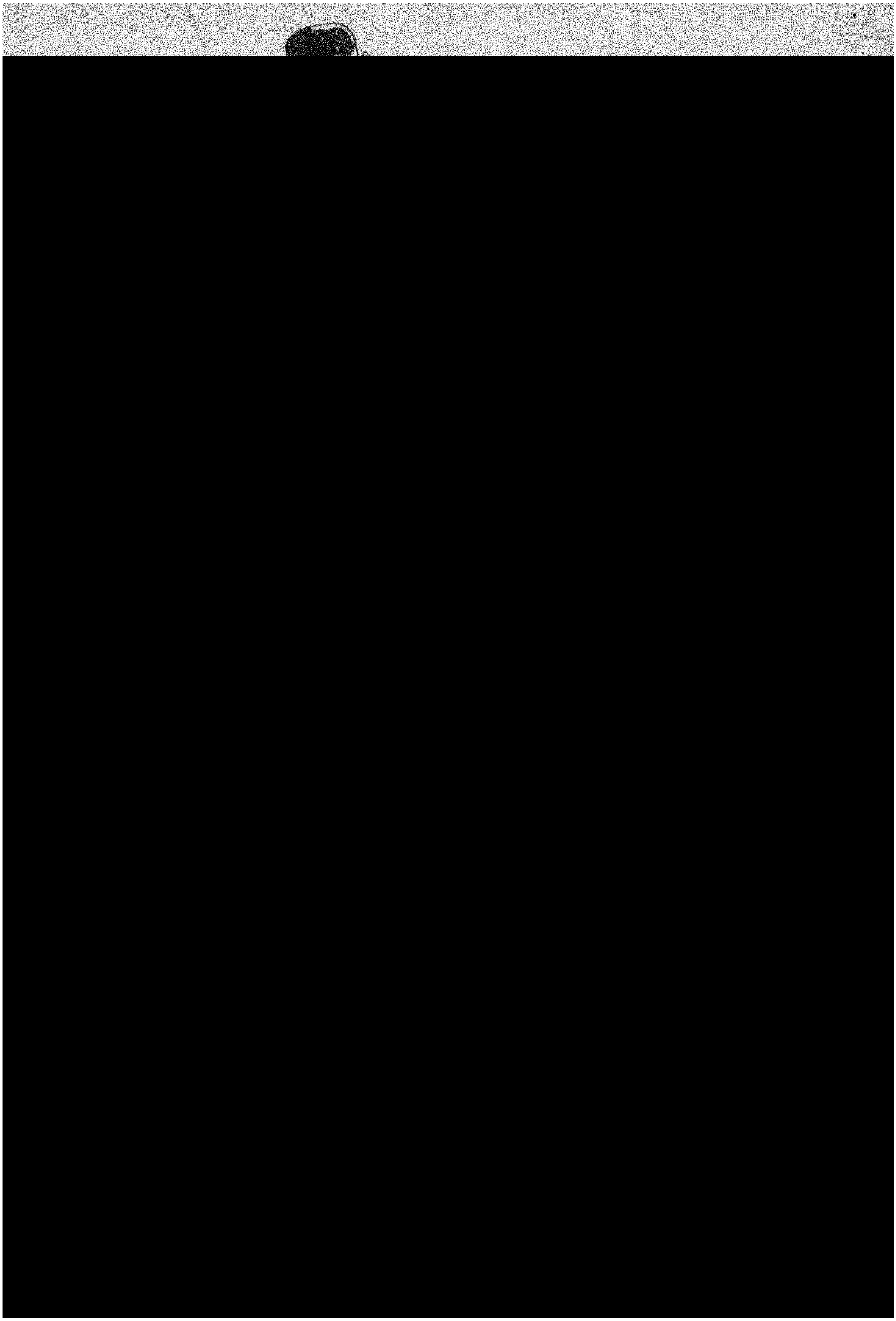

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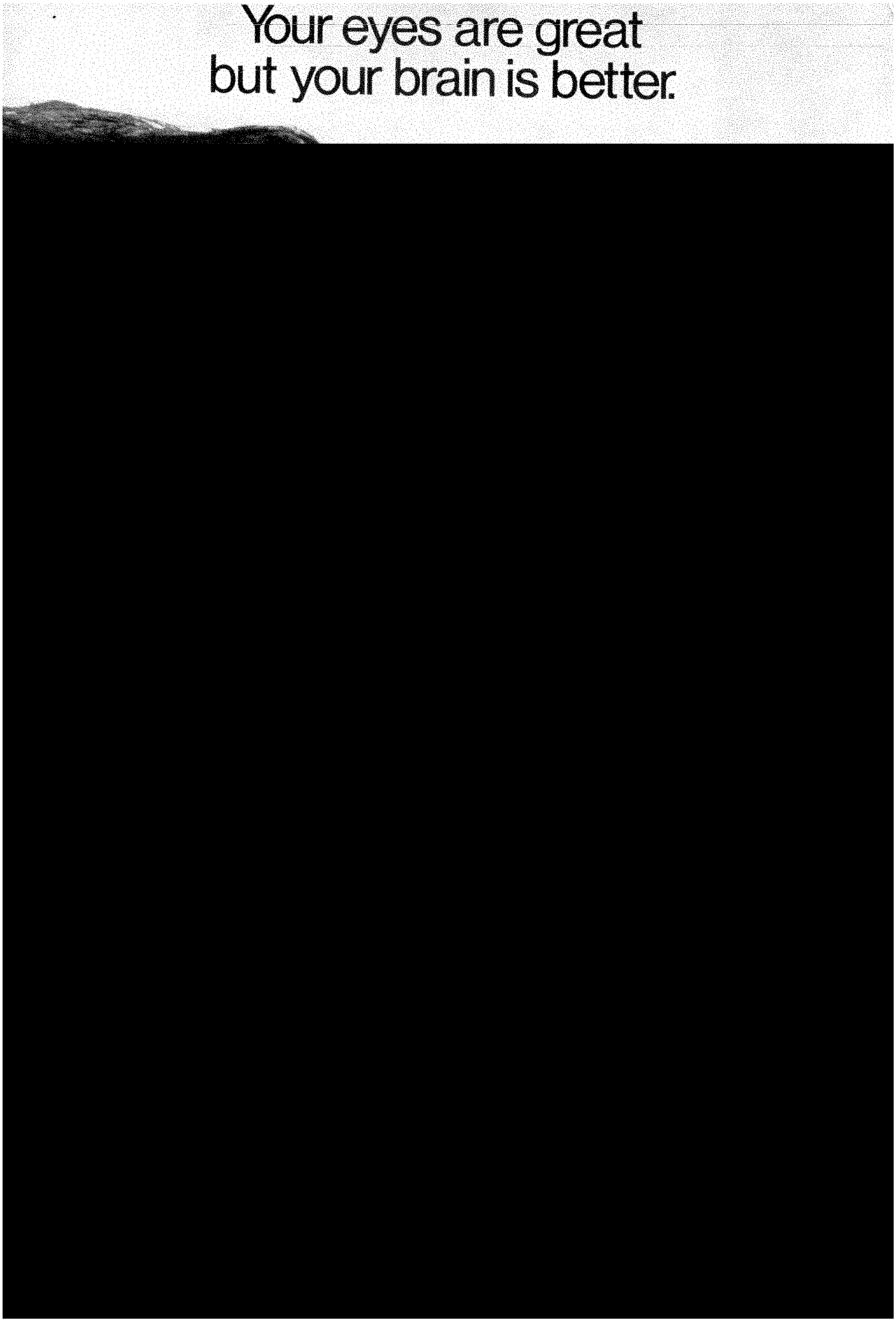
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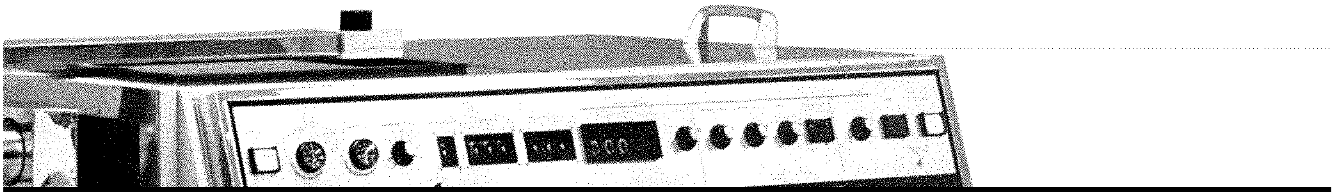

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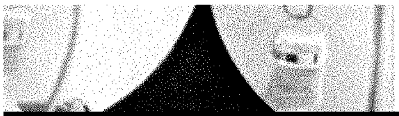

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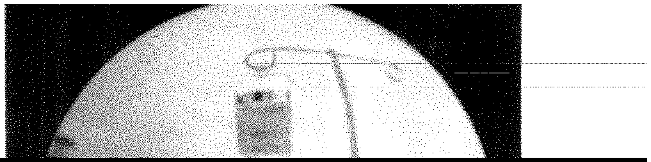

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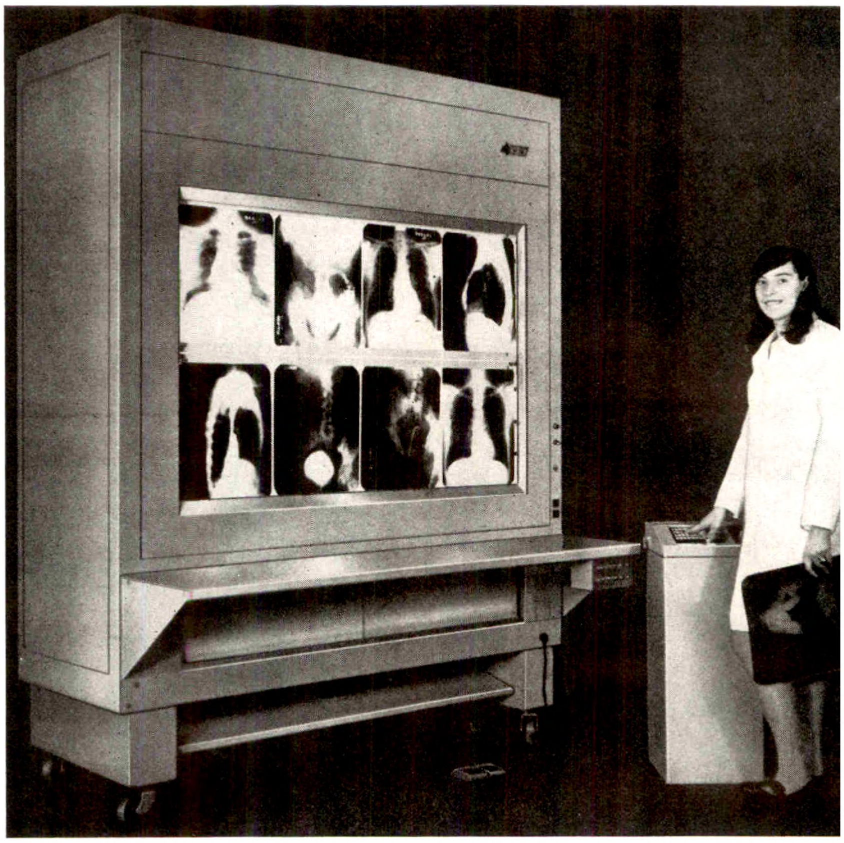

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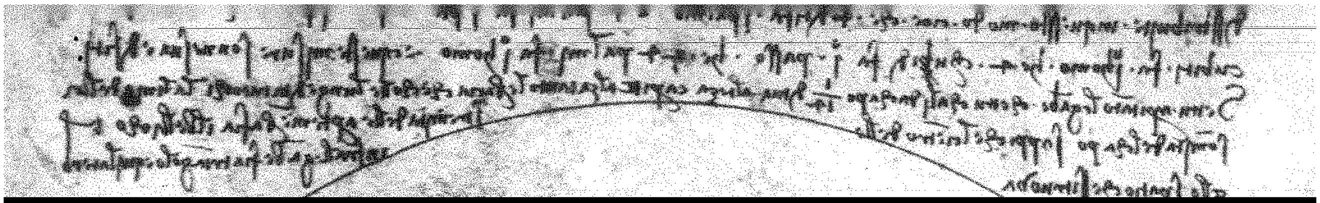

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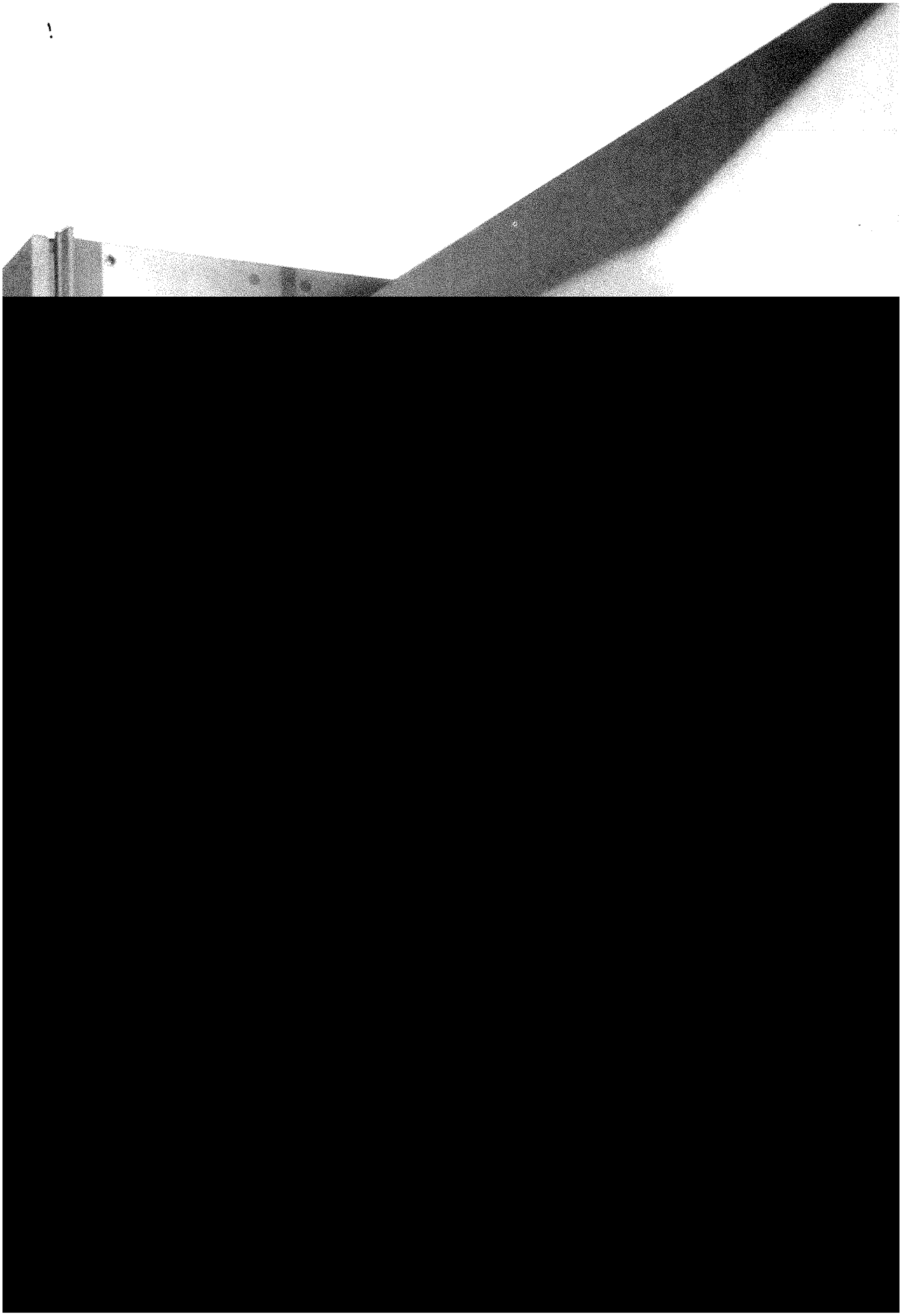

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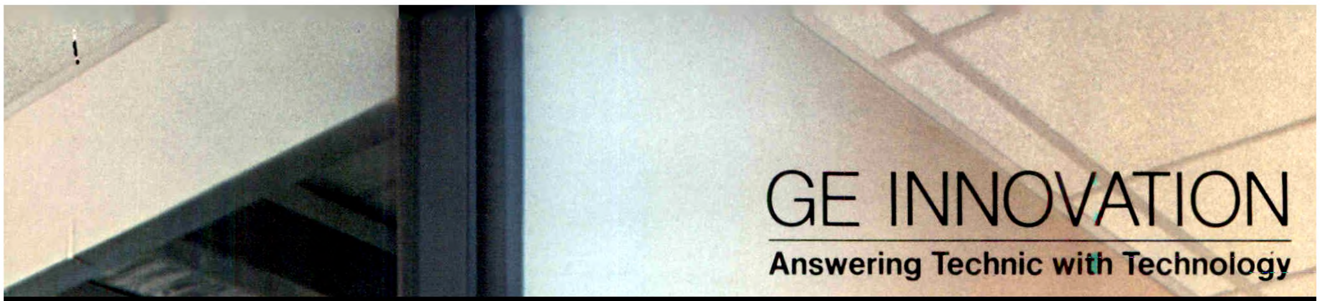

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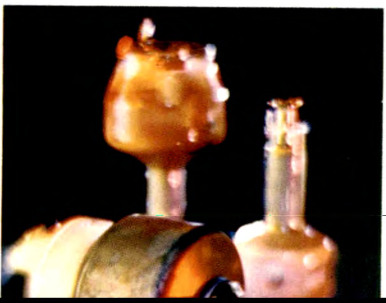

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By P. G. HESLIP, M.D., *and* T. R. OVERTON, PH.D.
EDMONTON, ALBERTA, CANADA

DURING the past 2 years we have studied dual radioisotope subtraction techniques for visualization of the pancreas. A gamma-ray camera and multiparameter analyzer equipped with digital magnetic tape were used for scan data acquisition and storage.

Image subtraction was carried out directly following a scanning procedure, using the 1,600 channel multiparameter analyzer and a cassette type magnetic tape recorder, thus providing an "immediate subtraction" image. Subsequently, a large scale digital computer was used for data smoothing, normalization and subtraction and for statistical evaluation of the image subtraction technique.

A detailed account of the experimental and analytic methods employed in this study will be reported elsewhere.[1] In the course of this investigation, 150 patients were examined and 160 pancreas scans obtained. Final diagnoses are available for 23 patients who presented with obstructive jaundice unassociated with biliary colic (17 patients), or with other symptoms suggestive of pancreatic malignancy (6 patients). The correlation of final diagnosis and scan interpretation for these 23 patients is presented in this paper. Several selected cases are illustrated to demonstrate the techniques used and types of scan images obtained.

Many cases presumed to have pancreatic malignancy on the basis of radiologic, clinical and scan evidence are not included in this series as pathologic corroboration was not available.

MATERIAL

A. CARCINOMA OF THE PANCREAS

Eight patients who presented with obstructive jaundice were subsequently proven to have carcinoma of the pancreas. The pancreas scan was abnormal in all 8 cases. Five scan images showed localized defects (*e.g.* Case I and II) and 3 showed generalized decreased and irregular radioisotope uptake (*e.g.* Case III).

CASE I. A 67 year old male investigated for cervical lymphadenopathy proven to be met-

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astatic anaplastic carcinoma showed no primary lesion on complete respiratory, gastrointestinal and renal investigation.

A pancreas scan (Fig. 1) was abnormal showing decreased and irregular radioisotope concentration in the head of the pancreas. A carcinoma of the cecum was suspected and a lower abdominal laparotomy performed, but no abnormality was found. The patient became jaundiced 4 months after the pancreas scanning and a percutaneous transhepatic cholangiogram in association with hypotonic duodenography suggested neoplastic involvement of the distal choledochus. At laparotomy carcinoma of the head of the pancreas was found.

Case II. A 49 year old male was investigated for vague upper abdominal pain and the gradual onset of obstructive jaundice and ascites. A routine upper gastrointestinal series was normal but a pancreas scan (Fig. 2) revealed widening of the porta hepatis in direct continuity with a localized area of decreased radioisotope concentration along the superior aspect of the head of the pancreas. The remaining pancreatic tissue showed relatively normal uptake of radioisotope. Abdominal laparotomy revealed carcinomatosis and ascites with extensive involvement of the porta hepatis and bile duct by carcinoma which also extended into the head of the pancreas. It was not determined with certainty whether the primary lesion arose in the common bile duct or in the head of the pancreas.

Case III. An 80 year old female developed abdominal pain. A duodenal ulcer was diagnosed, although no abnormality was found in the upper gastrointestinal series. The patient presented 6 weeks later with jaundice and fatigue.

An upper gastrointestinal series showed mucosal irregularity and rigidity in the postbulbar duodenum.

A pancreas scan (Fig. 3) demonstrated decreased and irregular uptake of radioisotope suggestive of extensive neoplastic disease or severe pancreatitis.

Laparotomy revealed a large carcinoma of the head of the pancreas.

B. CARCINOMA OF THE AMPULLA OF VATER, BILE DUCTS AND GALLBLADDER

Three patients presented with obstructive jaundice and with clinical symptoms suggestive of carcinoma of the pancreas. In all cases the pancreas scan was normal in appearance.

Case IV. A 74 year old male had a 3 week history of painless jaundice associated with moderate anorexia and weight loss.

The pancreas scan (Fig. 4) was considered completely normal. Percutaneous transhepatic cholangiography combined with selective hypotonic duodenography demonstrated a small mass in the region of the ampulla, felt to represent a small ampullary carcinoma. At laparotomy a 2 cm. carcinoma of the ampulla of Vater was found.

Case V. A 58 year old male developed malaise, nausea and painless jaundice over several weeks.

Upper gastrointestinal examination showed evidence of extrinsic pressure on the duodenum without direct invasion of the duodenal mucosa and a pancreas scan (Fig. 5) was normal. At laparotomy a large adenocarcinoma of the bile ducts without involvement of the pancreas was found.

Case VI. A 72 year old female presented with vague right upper quadrant pain and progressive jaundice of 6 months' duration.

Routine gastrointestinal roentgenograms were normal but a percutaneous transhepatic cholangiogram revealed uniform dilatation of the ductal system up to the middle segment of the common duct where complete obstruction was encountered.

A pancreas scan (Fig. 6) showed no abnormality.

Laparotomy revealed inoperable carcinoma of the gallbladder with involvement of the extrahepatic biliary system but without involvement of the pancreas.

C. SERUM HEPATITIS

Case VII. A 67 year old male was admitted for investigation of painless obstructive jaundice of 2 weeks' duration.

A routine upper gastrointestinal series suggested carcinoma of the head of the pancreas but a pancreas scan appeared normal. Selective hypotonic duodenography was then performed and showed no abnormality.

The patient gave a vague history of "needles" for an illness several months previously. His SGOT was 2,100 units on admission. He was treated conservatively and his elevated bilirubin and SGOT resolved spontaneously over 3 weeks. The patient was discharged with a

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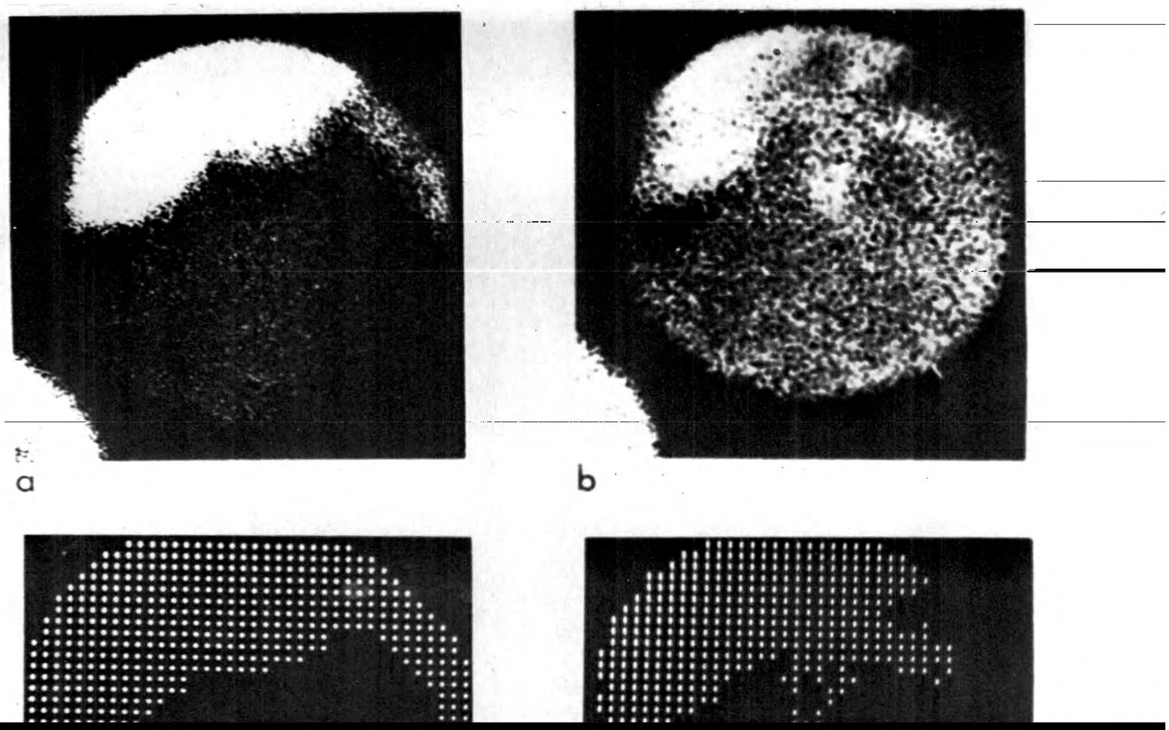

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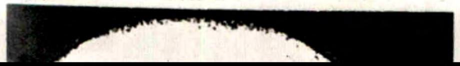

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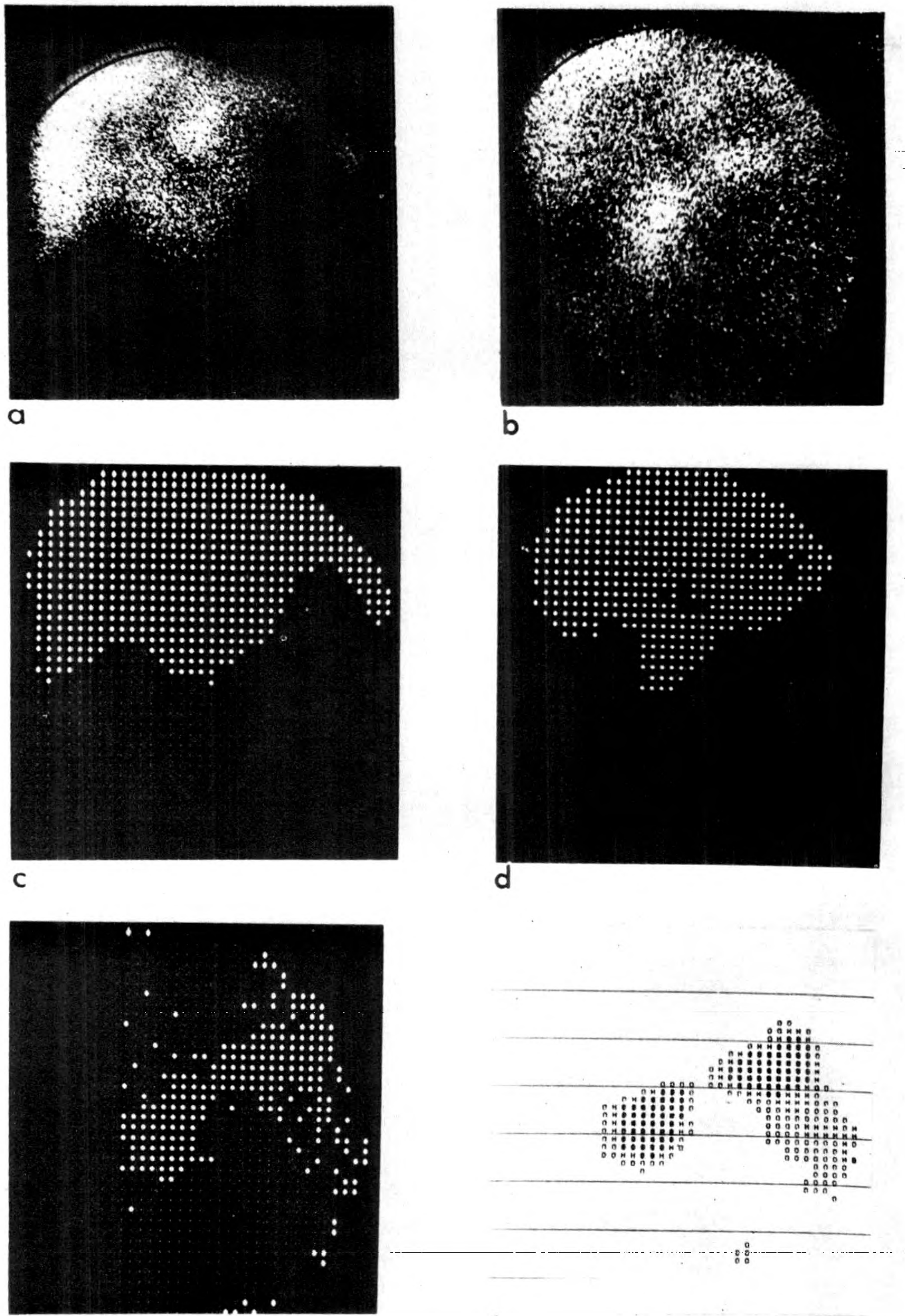
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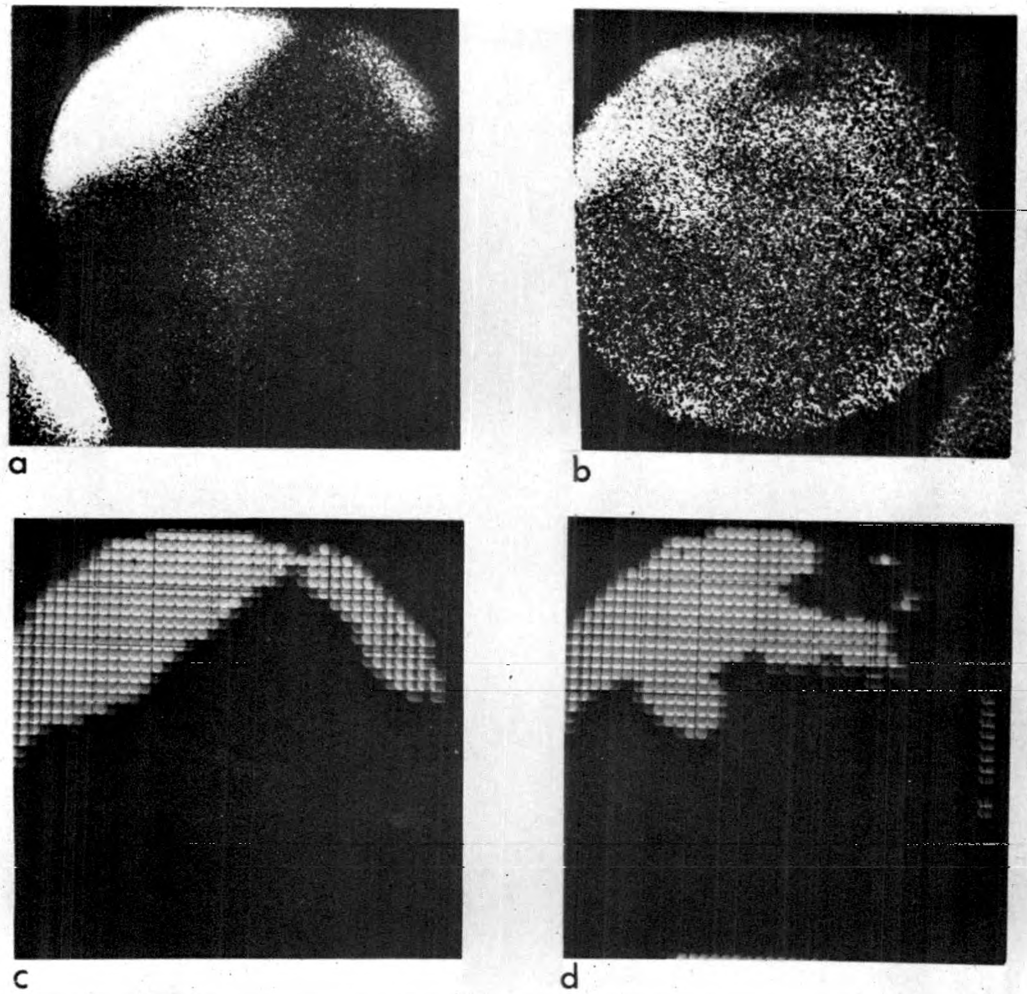
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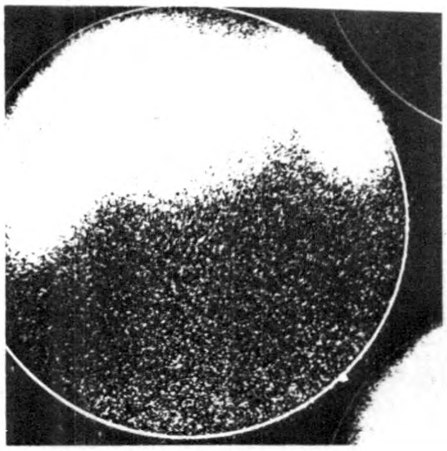

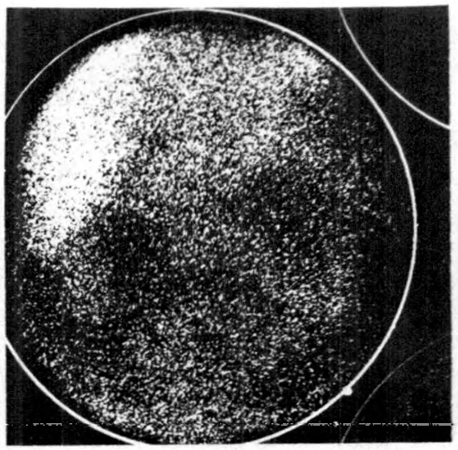

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DISCUSSION

Abnormal pancreas scans were obtained in 14 patients with proven carcinoma of the pancreas. Unfortunately, the scan appearance with carcinoma is not completely diagnostic, since chronic pancreatitis can produce the same type of scan abnormal-

(b) Increased blood supply to the pancreas related to decreased hepatic blood flow in obstructive jaundice is also possible, again providing more radioisotope to the pancreas.

It has been shown experimentally that only about 7.5 per cent of hepatic uptake

[redacted]

EVALUATION OF CORRECTIVE SURGERY FOR PANCREATITIS BY SE^{75} SELENOMETHIONINE PANCREATIC IMAGING[*]

A CASE REPORT

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THE use of pancreatic scintigraphy as a method for detecting pathology such as carcinoma, pseudocyst, and pancreatitis has been thoroughly described. While there is some disagreement as to the over-all value of this procedure, many authors[1,3,7] have endorsed it, with varying reservations, as a useful addition to the armamentarium available for investigation of a diagnostically refractory organ. Not so widely appreciated is the potential value of Se^{75} selenomethionine imaging as a means of documenting the return of pancreatic function to normal after an attack of acute pancreatitis or after corrective surgery for chronic pancreatitis.

This report describes a case in which the feasibility of such employment is demonstrated.

REPORT OF A CASE

A 40 year old Caucasian female presented with a 5 year history of intermittent upper abdominal pain. The initial attack had been of intense severity, but subsequent episodes had been rather minor until about 8 months prior to her admission. Thereafter, episodes of severe mid-epigastric discomfort with radiation to the back had occurred with increasing frequency. There was no history of nausea, vomiting, weight loss, fever, icterus, dark urine or light-colored stools. Although the patient drank moderately she was not considered an alcoholic.

Physical examination revealed minimal epigastric tenderness, but was otherwise negative. The serum bilirubin, alkaline phosphatase and fasting blood sugar values were all normal as were oral cholecystography and an upper gastrointestinal barium examination, but after administration of Se^{75} selenomethionine there was almost no identifiable accumulation of radioactivity by the pancreas (Fig. 1). In conjunction with the other findings this was taken to indicate pancreatitis and an evocative morphine prostigmine test[6] was performed. Baseline serum values for amylase and lipase were 14 Russell units and 1.2 cc., respectively. After parenteral administration of morphine (10 mg.) and prostigmine (1 mg.) the serum amylase rose to 68 Russell units and the lipase to 5.16 cc. During the test the patient experienced abdominal discomfort which duplicated her clinical symptoms. These results were considered clearly positive.

The patient was discharged with the diagnosis of chronic relapsing pancreatitis and treated medically with a low fat diet and anticholinergics. During the succeeding 6 months her symptoms continued. She experienced increasing epigastric discomfort which was exacerbated by alcohol. Accordingly she was re-admitted for attempted surgical amelioration. At celiotomy the pancreas was swollen and indurated and an intraoperative pancreatogram revealed filling of the secondary radicals, suggesting pancreatitis and low grade pancreatic ductal obstruction. A sphincterotomy and sphincteroplasty were therefore performed. A periampullary biopsy taken at this time was interpreted as showing fibrosis and chronic inflammation.

The postoperative course was uneventful, but a pancreatic scintigram obtained 2 weeks after operation showed little change from the preoperative study. During the succeeding

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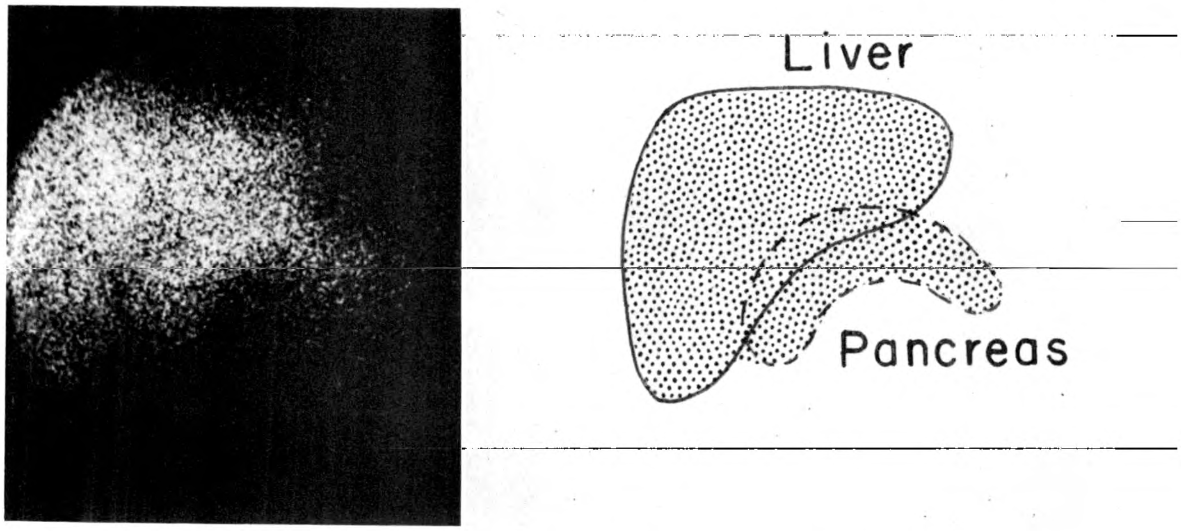

FIG. 2. Essentially normal accumulation of Se^{75} selenomethionine by the pancreas.

inactive in protein synthesis, and areas of such involvement will be permanently devoid of significant Se^{75} selenomethionine uptake. Acute pancreatitis, on the other hand, can transiently, but almost totally, inhibit pancreatic enzyme production even when no irreversible damage to the gland has

McCarthy and Brown[5] reported 3 patients in whom pancreatic scans were normal after recovery from acute pancreatitis, but the patient described in this report is, to the authors' knowledge, the first in whom pancreatic imaging returned to normal after well documented chronic relaps-

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preoperative Se^{75} selenomethionine pan-

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INTRAVENOUS RADIOISOTOPE ANGIOGRAPHY WITH COMPUTER PROCESSING OF DATA*

By MERLE K. LOKEN, M.D., Ph.D., RICHARD PONTO, B.S., ROBERT BACHE, M.D.,
and HOWARD BURCHELL, M.D.
MINNEAPOLIS, MINNESOTA

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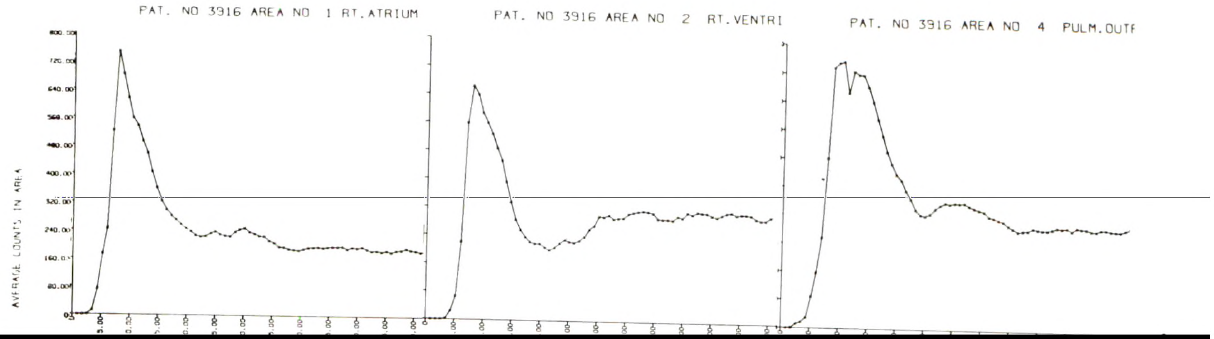
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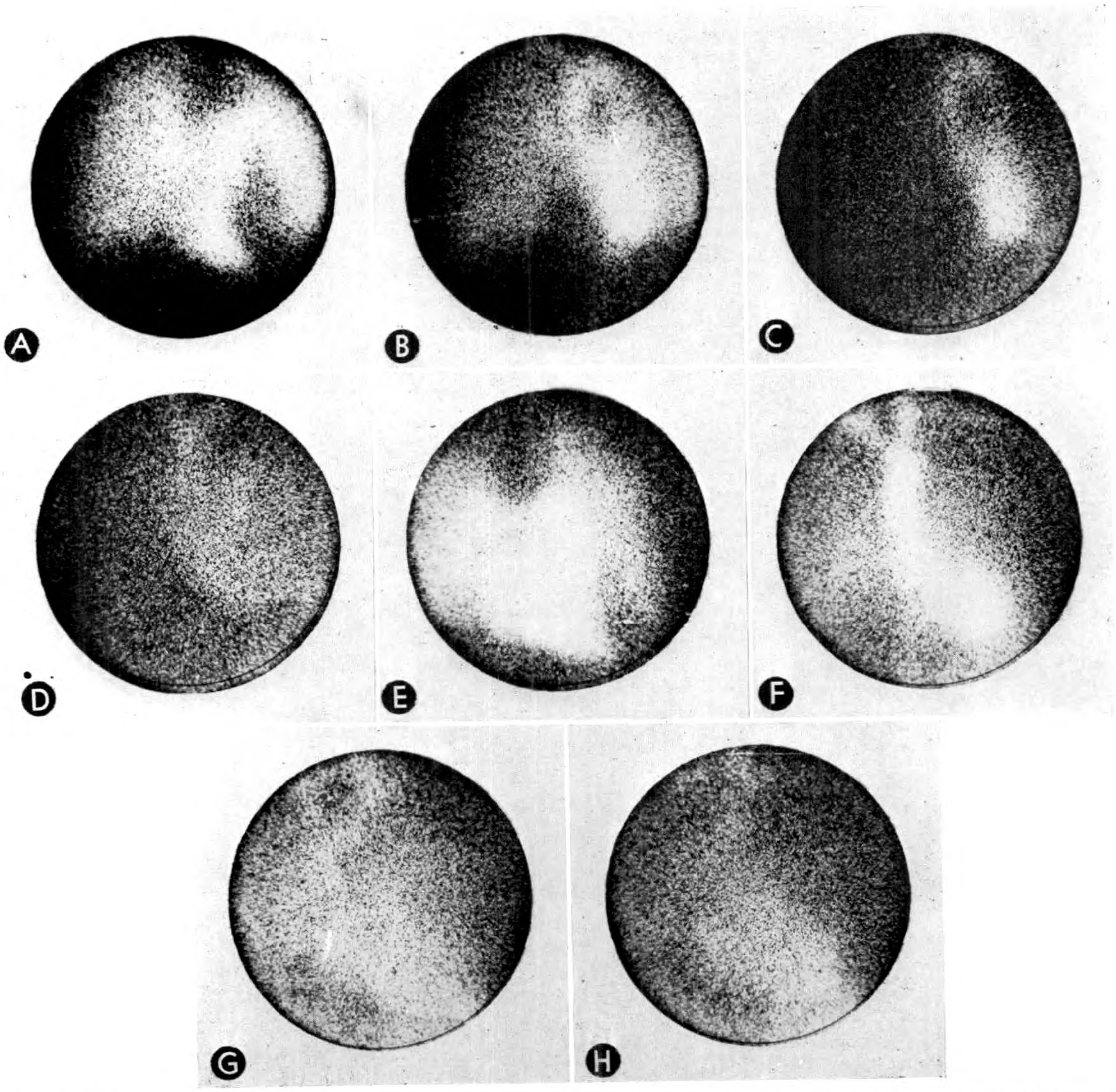

Fig. 3. Selected scintiphotograms showing the passage of Tc^{99m} (administered into the right atrium) through the cardiovascular system of a normal subject (J.J.) in the left anterior oblique position: (*A*) 2–4 seconds; (*B*) 4–6 seconds; (*C*) 6–8 seconds; (*D*) 10–12 seconds. The study was repeated in the anterior position: (*E*) 2–4 seconds; (*F*) 4–6 seconds; (*G*) 6–8 seconds; (*H*) 8–10 seconds.

activity through the heart and lungs. Selected Calcomp plots for areas representing the passage of radioactivity through the right atrium, right ventricle, pulmonary outflow tract, left lung, left ventricle and aorta are shown in Figure 5, *A–F*. The sharpness of the bolus in its passage through these areas is well demonstrated, with an indication of the recirculation of the radioisotope as a second peak which is

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Fig. 5. Computer plots of radioactivity *versus* time in selected regions of the cardiovascular system of subject (J.J.): (*A*) right atrium; (*B*) right ventricle; (*C*) pulmonary outflow tract; (*D*) left lung; (*E*) left ventricle; (*F*) aorta.

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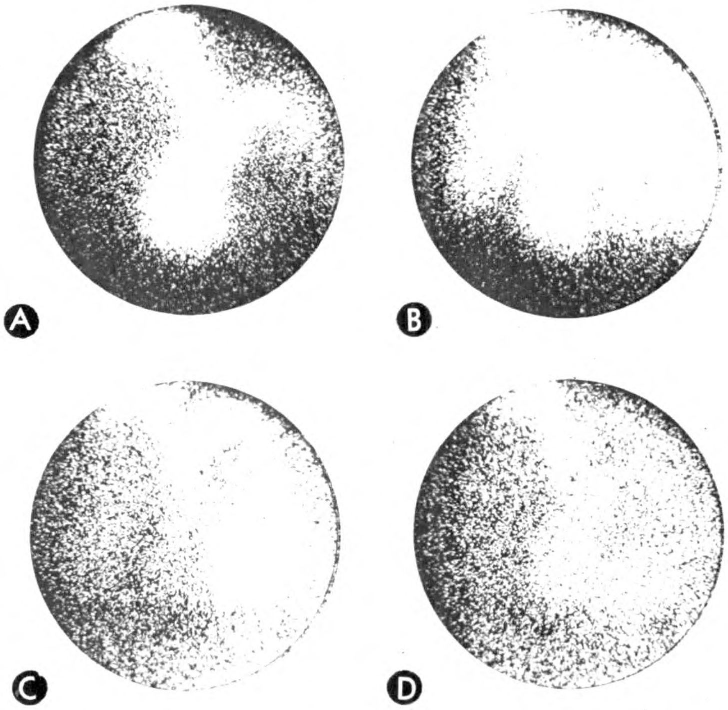

FIG. 6. Selected scintiphotograms obtained after intravenous administration of Tc^{99m} into a patient (L.J.) with heart disease: (*A*) 2–4 seconds; (*B*) 4–6 seconds; (*C*) 8–10 seconds; (*D*) 12–14 seconds.

considerably broader than the first because of dispersion of the bolus. The plot showing the sequential concentration of radioactivity over the pulmonary outflow tract shows a peak related to blood flow in the pulmonary artery followed by a second but lower plateau believed to denote pulmonary venous return.

CARDIAC DISEASE

The results obtained in the study of these normal subjects are in contrast with data from 2 patients with cardiac disease, one with mild and the other with more severe heart failure. Both were studied in the LAO position following the intravenous administration of 15 mc of Tc^{99m} as a bolus into an antecubital vein. In one (L.J.), selected scintiphotograms were obtained at approximately 2 second intervals, as shown in Figure 6, *A–D*. There is delayed emptying from the superior vena cava, as may be assessed from selected Calcomp plots (Fig. 7, *A–F*). The difference in contour contrasts with those from the normal subjects. The transition time from right ventricle to left ventricle is increased (peak times at 0.9 and 10.3 seconds, respectively). A more striking manifestation of distortion of the curves away from the normal occurs in the second patient (J.N.), who had severe pulmonary hypertension and was in cardiac failure. The heart was markedly enlarged (Fig. 8). Selected sequential scintiphotograms obtained following the technetium injection of this patient are shown in Figure 9, *A–F*. The timing of these studies is approximately the same (every 2 seconds) as that of previous illustrations. During this sequence of filming the radioactive indicator progresses very slowly from the enlarged right chambers of the heart into the pulmonary vessels. Selected Calcomp plots (Fig. 10, *A–E*) demonstrate the pronounced delay in passage of radioactivity through the cardiac chambers. The activity in the left ventricle reaches a plateau at only 70 seconds

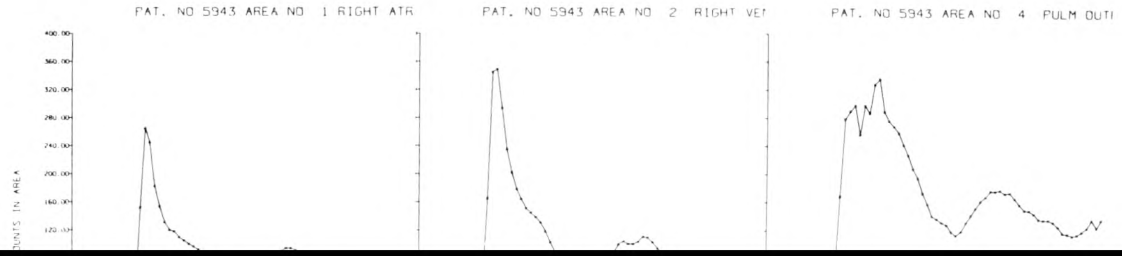
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Table I

CIRCULATION TIMES OBTAINED FROM THE COMPUTER

Patient No.	(S.R.) 3916		(J.J.) 3529		(L.J.) 5943		(J.N.) 8726	
Site of Tc^{99m} Bolus	Bolus Time (sec.)	Circulation Time (sec.)	Bolus Time (sec.)	Circulation Time (sec.)	Bolus Time (sec.)	Circulation Time (sec.)	Bolus Time (sec.)	Circulation Time (sec.)
RA	0	17.2	0	15.4	0	23.3	0	> 70
RV	2.1	17.3	1.7	15.5	0.9	26.7	1.7	> 70
PA	2.5	17.3	1.7	16.3	2.6	24.1	9.1	> 70
LL	4.3	16.4	3.5	15.5	4.8	24.5	12.1	> 70
RL	4.9	18.6	3.9	16.4	4.3	24.1	—	> 70
LV	7.1	16.4	6.9	15.9	10.3	23.3	19.1	> 70
Aorta	7.7	17.4	7.1	14.6	11.2	24.1	70	> 70
RV→LV	5.5		5.2		9.4		17.4	
Average		17.1		15.6		24.1		> 70

RA = right atrium; RV = right ventricle; PA = pulmonary artery; LL = left lung; RL = right lung; LV = left ventricle.

recirculation times is based on an average of the measured recirculation times to all areas selected. For the normal subjects this time ranged from 15.6 to 16.9 seconds, whereas for the 2 other patients the average values were 24.1 and greater than 70 seconds, respectively.

SUMMARY

Four studies are considered to illustrate

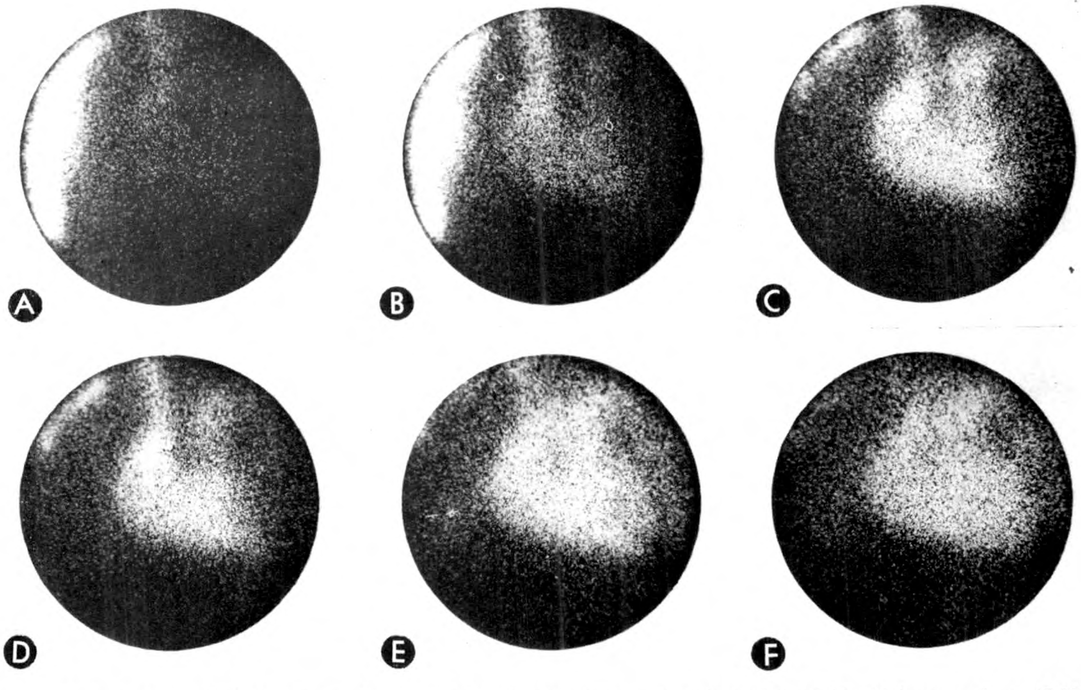

Fig. 9. Selected scintiphotograms obtained after intravenous administration of Tc^{99m} into patient (J.N.): (*A*) 4–6 seconds; (*B*) 10–12 seconds; (*C*) 14–16 seconds; (*D*) 20–22 seconds; (*E*) 26–28 seconds; (*F*) 38–40 seconds.

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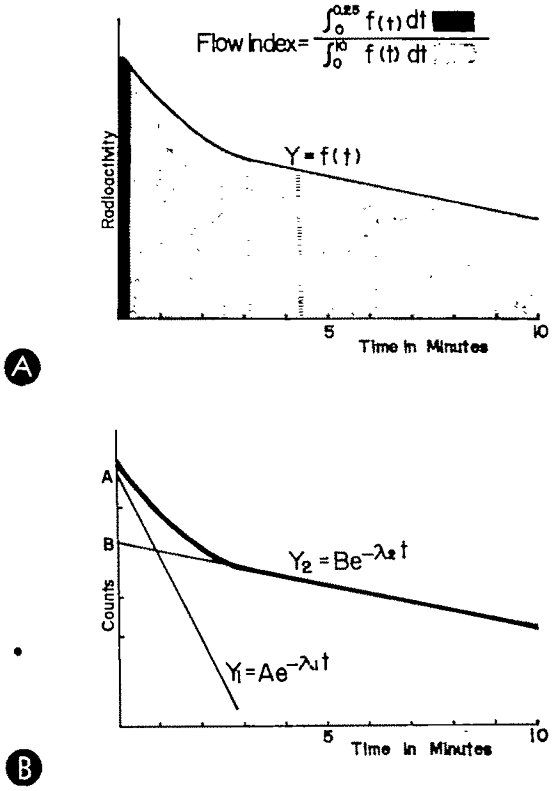

Fig. 1. Analysis of Xe^{133} washout curve. (*A*) Calculation of cerebral blood flow index in each element. (*B*) Compartmental analysis of the Xe^{133} washout curve.

The magnetic tape was fed into a digital computer for processing. To begin the analysis, the brain region was determined by cutting off the region with below 20 per cent of maximum radioactivities which was obtained by integrating all radioactivity from zero time to the tenth minute in each element of 1,600 matrices.

The cerebral blood flow index in each element of the brain region was calculated by the height/area method,[4,7,8,20,21] as demonstrated in Figure 1*A*. The height was determined by integrating the radioactivities from zero to the fifteenth second of the Xe^{133} washout curve, and the area under the curve was obtained by integrating all radioactivities from zero time to the tenth minute. Two points in X axis and 1 point in Y axis were intrapolated between 2 adjacent flow indices, and these values were divided into 8 levels. A map of the cerebral blood flow indices was printed out in a 90×40 array with a line printer, represented by codes, symbols and letters, as follows:

Level	*Symbol*
0	·
Below 44×10^{-3}	blank
45×10^{-3}– 59×10^{-3}	′
60×10^{-3}– 74×10^{-3}	″
75×10^{-3}– 99×10^{-3}	/
100×10^{-3}–149×10^{-3}	?
150×10^{-3}–199×10^{-3}	ø
Over 200×10^{-3}	#

In 3 cases, 100 mg. of nicotinamide was injected intravenously after 10 minutes of observation following the first injection of Xe^{133} into the internal carotid artery. Five minutes after the injection of nicotinamide, the second injection of Xe^{133} was performed according to the same procedure, and Xe^{133} washout curves were obtained from all the regions of the brain, keeping them in the same position.

Compartmental analysis of the Xe^{133} washout curve in the adjacent 2×2 elements in the brain region was performed[4,7,11,12,20] as demonstrated in Figure 1*B*. Two exponential functions, the fast component ($Y_1 = Ae^{-\lambda_1 t}$) and the slow component ($Y_2 = Be^{-\lambda_2 t}$), were obtained. *A* and *B* indicate the size of the Xe^{133} pool at zero time in the fast and the slow component, respectively; λ_1 and λ_2 show the clearance rate of the fast and the slow component, respectively (λ_2 was determined by the method of least squares of the logarithm of the chronologic counts on each adjacent 2×2 elements 2 or 3 minutes after the injection of Xe^{133}).

Thus,

$$\lambda_2 = \left[\frac{\sum_i y_i t - \frac{1}{N}\left(\sum_i y_i \sum t\right)}{\sum t^2 - \frac{1}{N}\left(\sum t\right)^2} \right], \quad (1)$$

where y_i was the logarithm of the counts in each adjacent 2×2 elements of the matrix,

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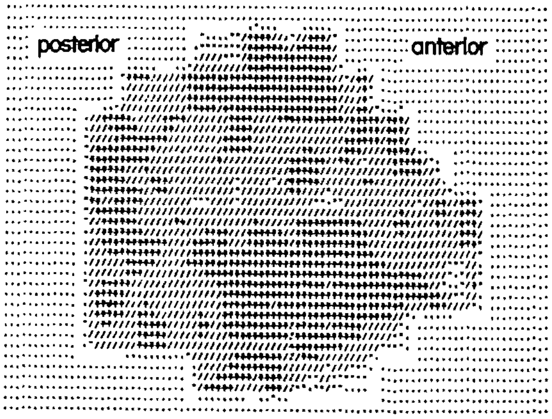

Fig. 3. Flow index map of a healthy 50 year old male.

the first injection of Xe^{133} were determined for the entire cerebrum.

RESULTS

(1). *Normal radioxenon brain scans.* Figure 2, *A* and *B* represents the findings in a healthy 26 year old female. Figure 2*A* shows the serial right lateral brain scintiphotos following the injection of Xe^{133}. A rapid decrease in radioactivity was observed over the entire cerebrum. Figure 2*B* shows a flow index map represented by 8 levels, depending on the degree of flow index in each element for the entire cerebrum. Regions with a relatively higher flow index were observed in the peripheral zone of the cerebral hemisphere and in the region of the insula or basal ganglia.

Figure 3 shows a flow index map for the entire cerebrum in a healthy 50 year old male. Almost the same results were obtained as in Figure 2*B*.

(2). *Abnormal radioxenon brain scans.* Figure 4 shows the findings in a 63 year old male with arteriosclerotic Parkinson's disease, exposed from the right side. Flow indices of the entire cerebrum showed lower values. The higher flow indices in the peripheral zone of the brain and in the region of basal ganglia, which were noted in healthy subjects, were not observed in this patient.

Figure 5, *A* and *B* shows the findings in a 44 year old male with hyperthyroidism. Rapid disappearance of Xe^{133} in the entire cerebrum is shown in Figure 5*A*. In a flow index map (Fig. 5*B*), higher indices were observed for the entire cerebrum.

Figure 6, *A* and *B* shows the findings in a 41 year old female with hypothyroidism. Slow disappearance of Xe^{133} in the entire cerebrum is shown in Figure 6*A*. In a flow index map (Fig. 6*B*), relatively lower indices were observed for the entire cerebrum.

Figure 7, *A* and *B* shows the findings in a 29 year old male with hydrocephalus following tuberculous meningitis. The pneumoencephalogram shows a huge hydrocephalus (Fig. 7*A*). Neurologic examinations disclosed no significant abnormalities. Figure 7*B* shows a flow index map from the right side. Abnormal distributions of the flow indices were observed, but their levels were within the normal range in the entire cerebrum.

Figure 8, *A* and *B* is from a 69 year old male with cerebral thrombosis. Radioxenon brain scintiphotos show a large area with poor perfusion in the right frontal region (Fig. 8*A*). In a flow index map, low indices were observed in the entire cerebrum, especially in the right frontal region (Fig. 8*B*).

(3). *Effect of "nicotinamide" on the Xe^{133} washout curve.* Figure 9, *A*, *B* and *C* shows maps of the ratios of $A\lambda_1$ of the fast component after the second injection to that after the first injection, and of $B\lambda_2$ of the slow component after the second injection to that after the first injection of Xe^{133} in

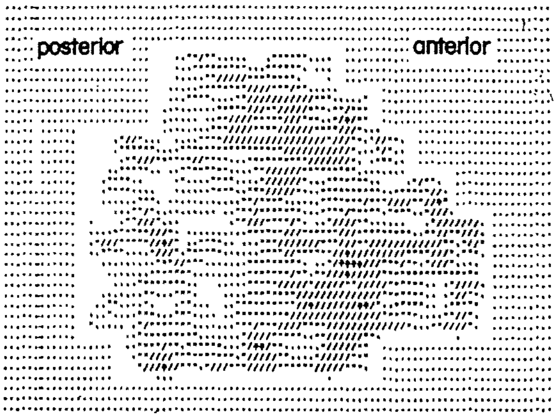

Fig. 4. Flow index map of a 63 year old male with arteriosclerotic Parkinson's disease.

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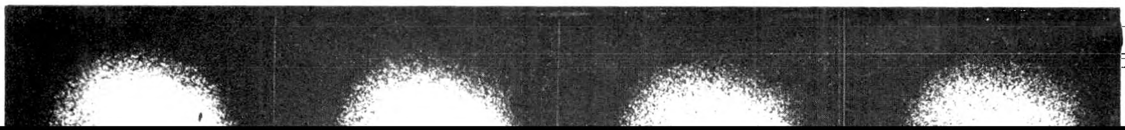

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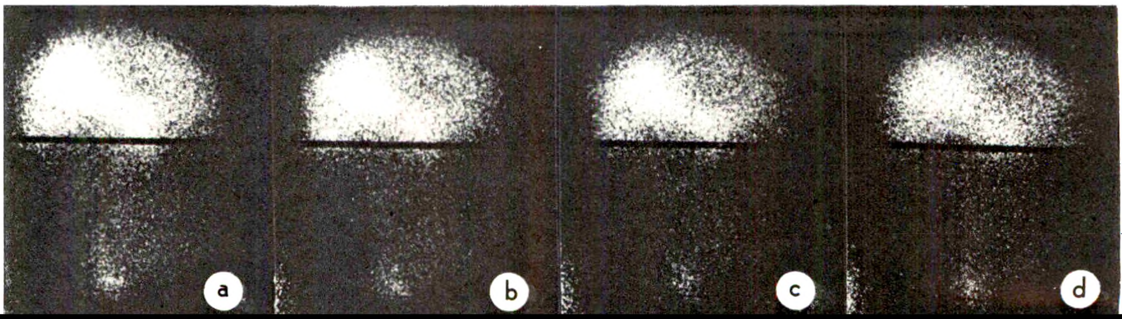
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CLINICAL APPLICATION OF THE BROAD SPECTRUM SCANNING AGENT—INDIUM 113M*

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THE clinical application of radiopharmaceuticals to assess body functions

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sults in an organ specific sterile and pyrogen free radiopharmaceutical. Thus far, we

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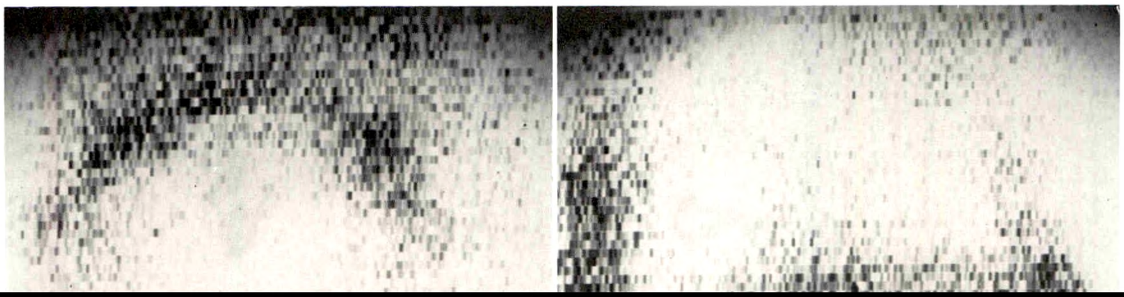

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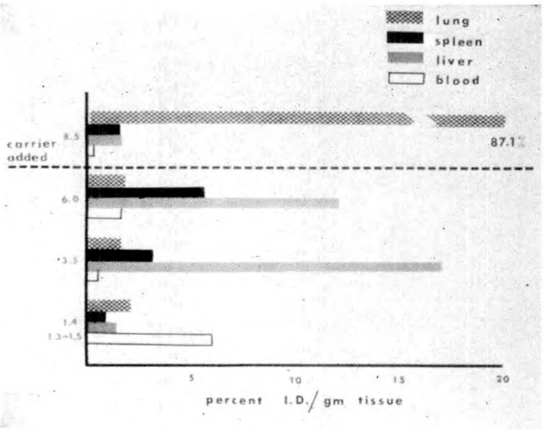


Fig. 5. The effect of pH on organ specificity in the rat. Increasing pH increments are obtained with the phosphate buffer described in the text. The lung preparation requires the addition of carrier to obtain the larger particles.

that as the pH increases with phosphate buffer, there is an increased deposition first in the liver and then in the spleen (Fig. 5). This distinction between liver and spleen was not as readily apparent when using hydroxide to elevate the pH. We have, therefore, used phosphate buffer ($Na_2HPO_4 \cdot 7H_2O$ 76.1425 gm., $NaH_2PO_4 \cdot H_2O$ 4.6030 gm. in 500 ml. pyrogen free sterile water, pH 7.4) to bring the eluant to a pH of 3.5 (0.9 cc.) which results in clinically useful liver scans. Similarly by increasing the pH slightly more there is an increased deposition in the spleen, but this distinction has not been clinically significant and a pH of 3.5 to a pH of 6 will result in adequate colloid reticuloendothelial scans (liver, spleen and bone marrow) (Fig. 6; 7; and 8).

Chelates for brain and kidney scanning. Initially, one-step preparation of DPTA and EDTA chelates using indium 113m as

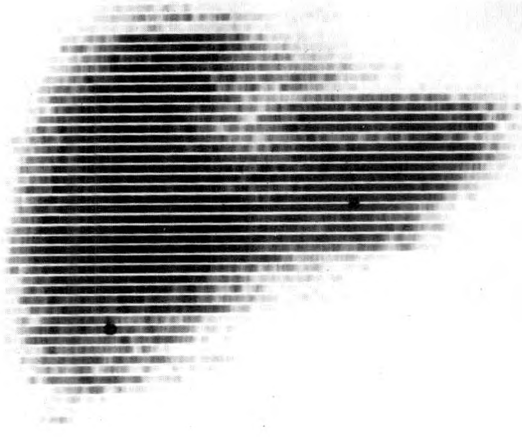


Fig. 6. Liver scan from a patient with proven liver abscess.

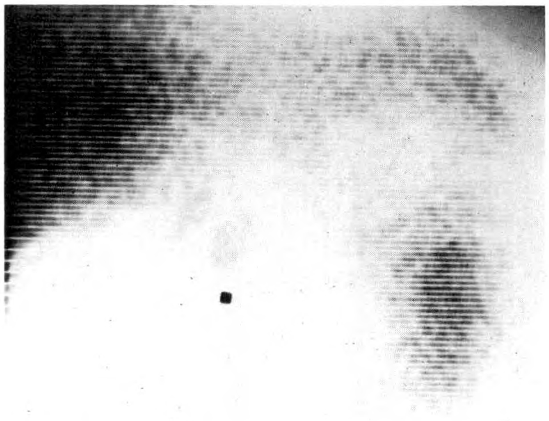


Fig. 7. Spleen scan from an 11 year old girl with a history of trauma. Filling defect in the mid-portion of the spleen was due to a subscapsular hematoma.

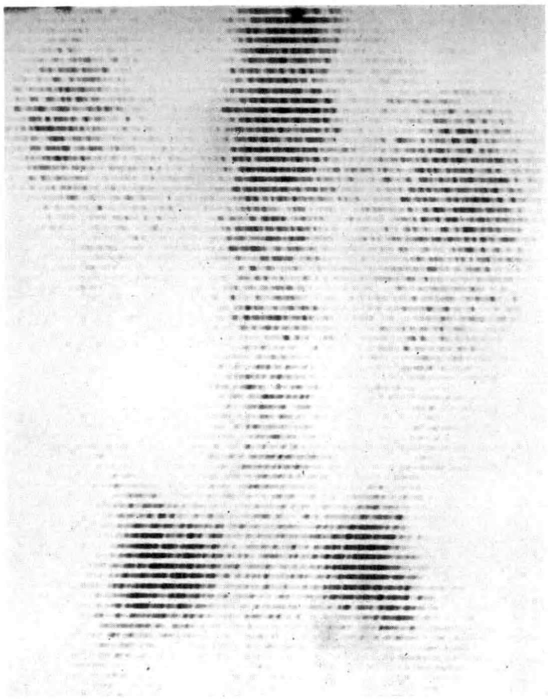
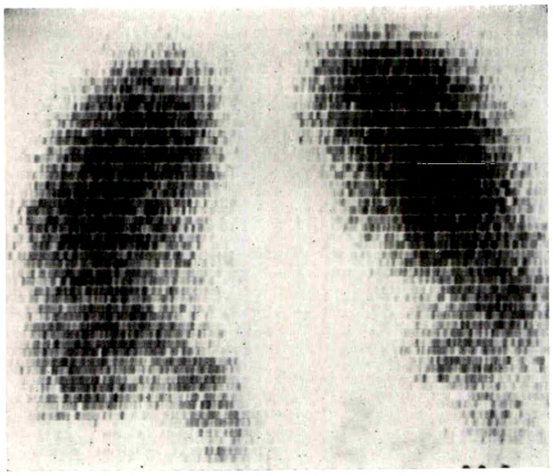


Fig. 8. Normal bone marrow scan of dorsal spine and pelvis from a patient with Hodgkin's disease.

eluted resulted in precipitation of the indium and reticuloendothelial deposition. Subsequent studies with an acetate buffer made a soluble indium acetate which is easily bound to DPTA.[2] This DPTA indium chelate (pH 7) is useful for brain and kidney scanning (Fig. 9, *A* and *B*).

Aggregates for lung scanning. A two-step preparation has been necessary for the formation of aggregates which are used for routine lung scans (Fig. 10). Preparation is as follows: 5 ml. indium 113m eluate is added into a vial containing 0.2 ml. $FeCl_3$ solution ($FeCl_3 \cdot 7H_2O$ 10 per cent). The content of this vial is transferred into a



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previous techniques in over 500 patients. The indium scans have sufficient photon yield to result in the same clinical utility as technetium. Thus, the theoretic considerations have not made a significant difference at the practical level. The distinct advantage of indium has been the ease of preparing organ specific radionuclides, which obviates the need for radiochemists and more sophisticated manipulation of the radioactive pharmaceutical. One-step preparation allows for the widespread availability of this short-lived scanning agent in most centers using clinical radioisotopes.

SUMMARY

Indium 113m is a transition metal obtained by eluting a tin 113 generator system with HCl. The complexing ability of the eluted indium allows for one-step chemical preparation of organ specific scanning agents. Indium transferrin is used for plasma volume estimation and cardiac blood pool-placental scanning; indium phosphate colloids are used for reticuloendothelial scanning, *i.e.*, liver, spleen and bone marrow; indium DPTA chelates are used for brain and kidney scanning; and indium-iron hydroxide macroaggregate is used for perfusion lung scans. Clinical experience in over 1,000 patients suggests that these agents are at least as effective as previous scanning radiopharmaceuticals.

The short half life of the indium (100 minutes) and the long half life of the tin (118 days) allow for frequent elution of the generator in a clinical setting. This markedly decreases cost and obviates the need for storage of various isotopes at varying decay rates. The one-step process also eliminates the need for sophisticated radiochemical preparation prior to each scan.

This availability of an inexpensive broad spectrum scanning agent may permit a more widespread application of diagnostic nuclear medicine procedures where they are now not feasible.

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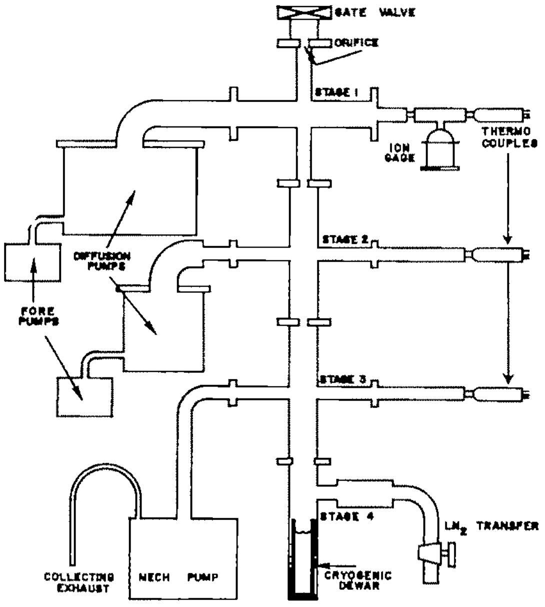

Fig. 1. Cross section of vacuum gradient system.

withstand the heat of bombardment. It would certainly rupture.

To permit use of a relatively low energy particle beam and a gaseous target, a new type of window using differential, graduated vacuums was devised. This represents, in a sense, an "empty window" instead of the conventional metal ones.

METHOD

The vacuum gradient system consists of 4 separate but communicating chambers or stages. These stages are separated from one another by metal diaphragms, in each of which there is a hole. These orifices are aligned, and it is through them that the accelerated particle beam passes (Fig. 1).

The topmost stage is coupled to the accelerator portal, with a gate valve immediately above the connection. This valve is opened only when the vacuum in the gradient system is of the same order as that in the accelerator. The upper surface of the top stage (Stage 1) has a 4 mm. orifice communicating with the small space between it and the gate valve. The inferior surface of Stage 1 consists of a plate containing another 4 mm. hole leading into the body of the second stage. In the same way, the bottom plate of Stage 2 communicates with Stage 3 through another 4 mm. opening.

Stage 4 is completely open to Stage 3 above it. It contains a cryogenic Dewar which can be filled from outside with liquid through a valve without dismantling the system. Atmospheric air or other gasses can also be leaked into Stage 4 through this valve under controlled conditions if desired.

Stage 1 is evacuated by a 6 inch diffusion pump supported by a suitable mechanical pump. The pressure in this stage is monitored with an ionization gauge.

Stage 2 is evacuated by a 4 inch diffusion pump also working with a mechanical pump. The pressure in this chamber is measured using a thermocouple previously calibrated against a McLeod gauge.

Stage 3 is evacuated only by a high speed mechanical pump. The pressure at this level is measured with another thermocouple.

Since our major interest was the potential result of bombarding nitrogen with a deuteron beam, the system was tested with the cryogenic Dewar in Stage 4 filled with liquid nitrogen. The problem was to evacuate the system to a level at which it would be possible to allow the deuteron beam to enter Stages 3 and 4, which together would constitute the reaction chamber.

With the gate valve closed, the mechanical pumps were started. After about 15 minutes of pumping, the pressures in Stages 1 and 2 were sufficiently low to permit the diffusion pumps to be heated. After an additional 20 minutes, the pressure in Stage 1 was about 1×10^{-5} mm. Hg, in Stage 2, 5×10^{-3} mm. Hg, in Stage 3, 2×10^{-1} mm. of Hg. At this point, the gate valve to the accelerator was opened. The ionization gauge in the accelerator showed no increase in pressure above the useful range of 9×10^{-6} mm. of Hg, the difference between the pressure in Stage 1 and accelerator being handled by the pumping system of the accelerator itself. These con-

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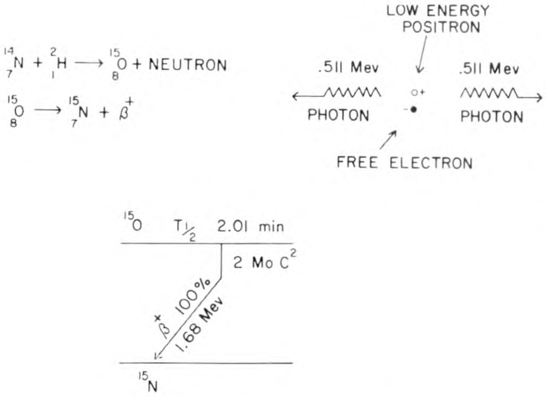
$^{14}_{7}N + ^{2}_{1}H \longrightarrow ^{15}_{8}O$ + NEUTRON
$^{15}_{8}O \longrightarrow ^{15}_{7}N + \beta^+$
LOW ENERGY POSITRON
.511 Mev
.511 Mev
PHOTON
PHOTON
FREE ELECTRON
^{15}O $T_{1/2}$ 2.01 min
2 Mo C^2
β^+ 100%
1.68 Mev
^{15}N

Fig. 3. Production and decay of oxygen 15.

The nitrogen target used by others has usually consisted of nitrogen gas with 4 per cent oxygen,[1] but simple room air has also been used.[8]

METHOD

The work presented here was done with a Van de Graaff generator, High Voltage Engineering Model PN 400, capable of accelerating deuterons to a maximum of 400 kev. We recognized that this energy level was not ideal, but this was the apparatus available to us.

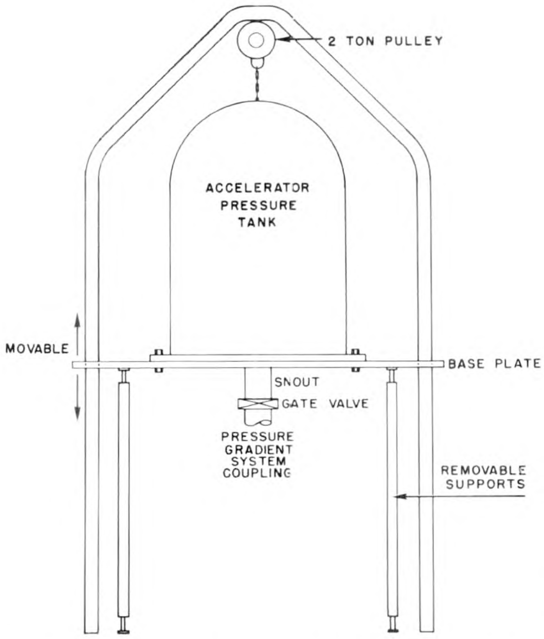

Fig. 4. Cross section of accelerator support structure.

The Van de Graaff, originally intended for horizontal operation, was suspended vertically (Fig. 4). At the time of installation, provision was made to allow the generator to be moved up and down. In addition, the shielding can be raised without moving the generator core, making it easier to service the instrument.

The snout of the Van de Graaff generator was coupled to the topmost stage of the differential vacuum system, with the gate valve immediately above the connection, in the manner described in Part 1 above (Fig. 5) The cryogenic Dewar in Stage 4 was filled with 1 liter of liquid nitrogen.

After the liquid nitrogen has been transferred into the Dewar, the vacuum system is flushed with gaseous nitrogen for a few minutes to remove any residual air or water vapor. The external connection to the Dewar is then closed to the atmosphere, and the mechanical pumps are started. The diffusion pumps are heated after about

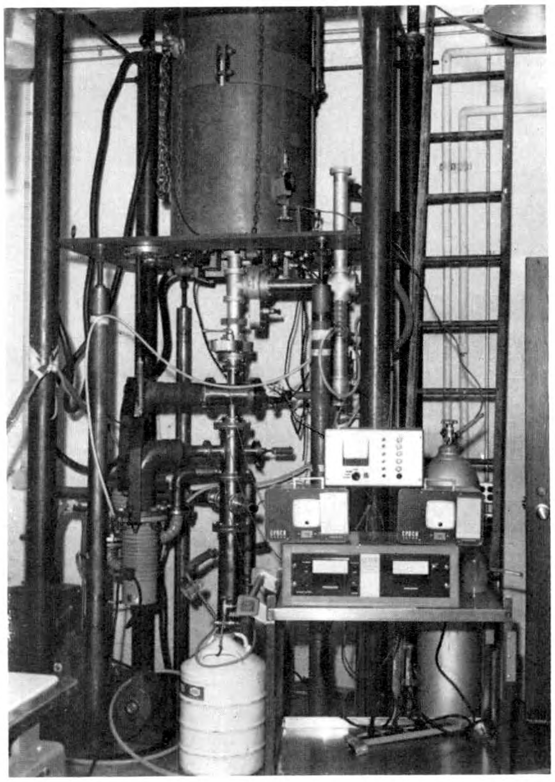

Fig. 5. Photograph of Van de Graaff generator and vacuum gradient assembly and related equipment.

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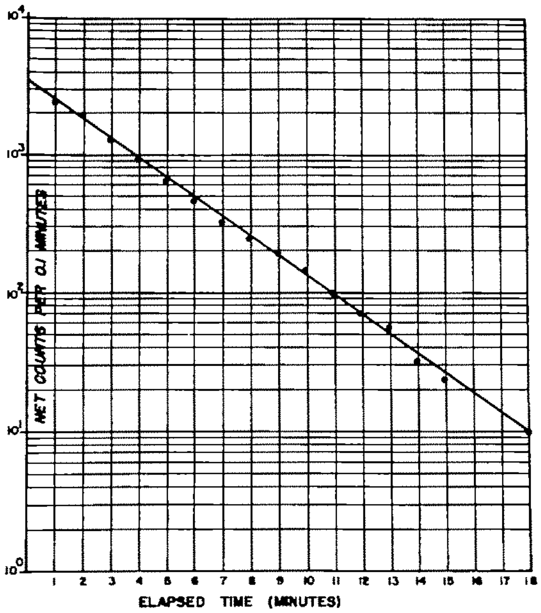

Fig. 8. Half life determination of 2 cc. sample of effluent gas ($T\frac{1}{2} = 2.1$ min.).

system. Curve B shows the actual growth in the dynamic system we used. The reasons for the difference are the large dead space in the mechanical pump, the time lag required for the gas to flow from the pump to the detection system (about 2 minutes), and an increase in the rate of evaporation of the nitrogen during the actual deuteron bombardment. The last produces an increase in the pumping speed of the mechanical pump causing a larger dilution of the O^{15} in the effluent.

By knowing the net counts per minute, the efficiency of the counting system, the rate of flow of the effluent gas, and the deuteron current reaching the reaction chamber, the quantity of O^{15} obtained was calculated. The activity produced under these conditions was 14 microcuries per liter of gas per minute per microampere of current reaching the reaction chamber.

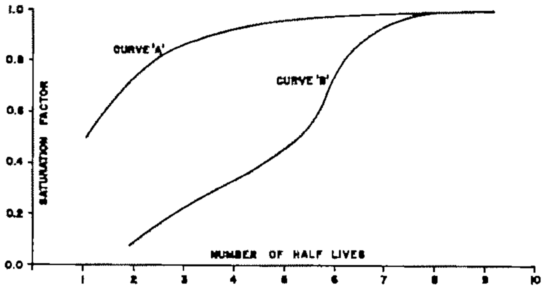

Fig. 9. Equilibrium curve of oxygen 15 in effluent gas. Curve A is theoretical; Curve B experimentally obtained.

DISCUSSION

We have shown that it is possible to produce O^{15} using a relatively low energy particle accelerator with an internal nitrogen target and a vacuum gradient system acting as a window for the deuteron beam. The quantities of the isotope we generated and its specific activity are not really in the clinically useful range. A substantially increased yield could be anticipated if the energy of the generator were somewhat higher, with an optimal level of about 2 mev. (Fig. 10).

However, even at the 400 kev. level at

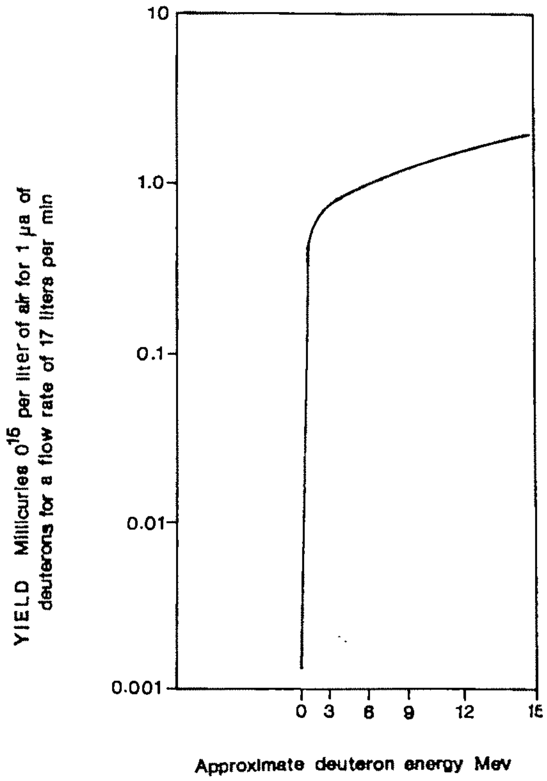

Fig. 10. Production of oxygen 15 as a function of deuteron energy (from Dyson *et al.*[5]).

which we have been working there are practical methods of increasing the yield. The production of O^{15} is directly related to the quantity of the deuteron beam reaching the reaction chamber. Because of the small size of the orifices between the upper stages of the vacuum gradient system, which were burned through by the beam itself, most of the beam was lost above the reaction chamber. We are currently devising a method of controlling both the sizes and positions of these holes to allow

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about 2 meters at −233°C. The dense cloud of gas and the solid nitrogen below it plus the decreased range of the deuterons at low temperature increase significantly the probability of an interaction.

Furthermore, both liquid and solid nitrogen have a relatively high heat capacity.[7] This allows the introduction of substantial quantities of heat during deuteron bombardment without an appreciable increase in temperature. In addition to these con-allowing the use of relatively low energy deuterons to bombard gaseous targets.

This system consists of 4 communicating chambers which can be evacuated to produce a high vacuum at the accelerator end with a poor vacuum at the level of the gaseous target. Using this device with a 400 kev. Van de Graaff generator and a nitrogen target, a significant quantity of oxygen 15 was produced.

With relatively minor modifications to the system, clinically useful quantities of the isotope can be obtained.

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We wish to thank the General Motors Research Laboratories Physics Department for their gift of the Van de Graaff generator used in this work.

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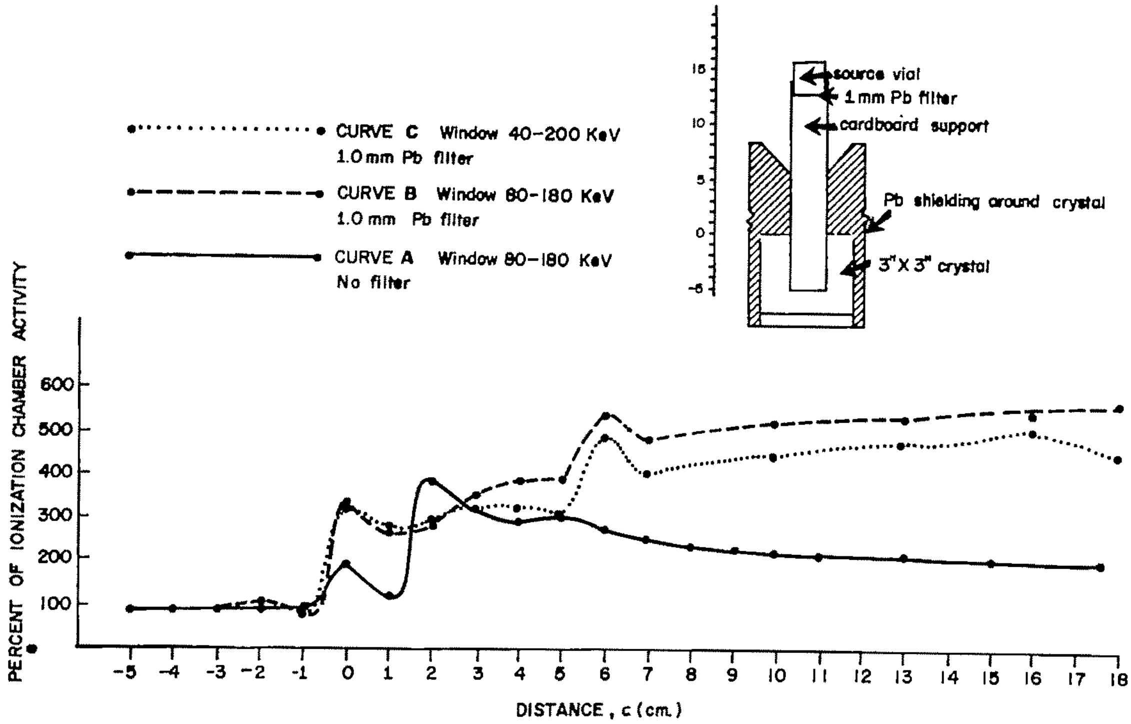

FIG. 1. Activity of Tc^{99m} expressed as a percentage of ionization chamber activity as determined using a Co^{57} standard with a well scintillation detector under a variety of conditions.

counts were made for decay and coincidence losses.

Activities are presented as a percentage of the activity determined by the dose calibrator. When activities were derived using a Co^{57} standard, the following equation was used[1]:

$$\text{Activity } (Tc^{99m}) = \frac{0.91 \times \text{cpm } Tc^{99m} \times \text{Activity } (Co^{57})}{\text{cpm } Co^{57}}.$$

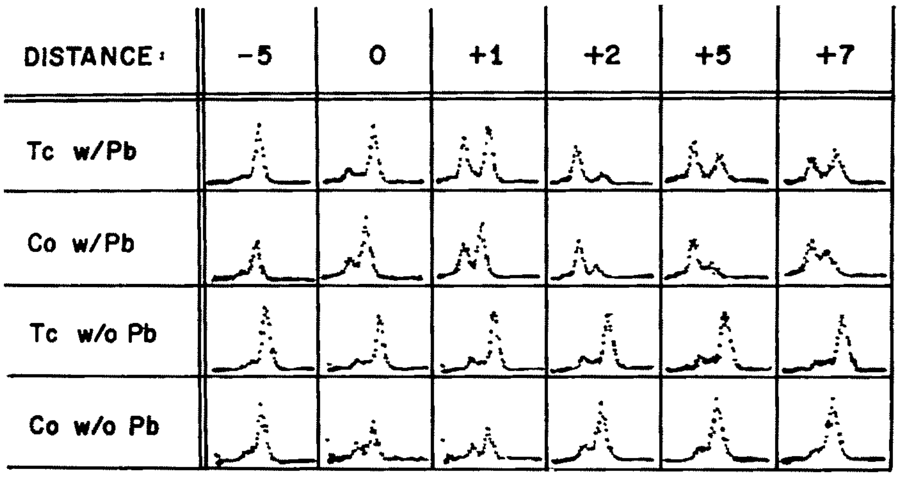

FIG. 2. Tc^{99m} and Co^{57} gamma spectra from 0 to 200 KeV. as a function of distance and filtration as obtained with a 100 channel analyzer and a shielded 3×3 inch NaI(Tl) crystal. Counting times sufficient to illustrate the spectra were employed.

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varying activities (95 to 570 per cent) are obtained depending on the geometry, filtration, and window setting employed. The spectra obtained with selected data points are shown in Figure 2.

C. *Calibration with a flat face crystal.* Figure 3 shows the results obtained from performing similar experiments with a flat face crystal. Values obtained without filtration (Curve D) were constant and approximately correct (88–99 per cent), independent of the distance at which the calibration was made. When the experiment was repeated using a 1 mm. lead filter and a window setting of 40 to 200 KeV., results obtained were erroneous, varying from 81 to 176 per cent of true activity (Curve E). Geometry greatly influenced the results obtained. When a lead filter which entirely covered the crystal face was used, completely erroneous results were obtained for all geometries (Curve F), with a range of 300 to 500 per cent.

DISCUSSION

Our results confirm that calibration in a well or on a flat face crystal without filtration gives approximately correct assays using the formula given above. This is in general agreement with the findings of Smith,[1] indicating that the assumption is valid that the counting efficiencies for Co^{57} and Tc^{99m} are practically identical ***under these conditions***. However, deviations from this method, as used by some manufacturers, wherein filtration or geometry is altered, may give markedly incorrect assays. The resultant degree of error is dependent on the particular filtration and/or geometry selected. This is because it is assumed that the relative counting efficiencies for Co^{57} and Tc^{99m} are unaffected by differences in geometry or attenuation. Many users and manufacturers are unaware that this assumption is invalid; the results illustrated in Figure 2 demonstrate this. It is obvious that spectral shifts occur with Co^{57} and Tc^{99m} as filtration is employed. Obvious shifts are also apparent without added filtration as the geometry is altered. This is due to the varying degree of scatter from crystal shielding which occurs as the distance and incident angle to this shielding are altered. This is illustrated by the rapid changes reflected in the curves shown in Figure 1, at which points the source enters the shielding surrounding the crystal and then the crystal itself. Thus, differences in scattering of the primary photons of the 2 radioisotopes and differences in absorption of the scattered photons by the crystal obviously account for differences in efficiency of counting; incorrect calibrations can occur if these factors are not taken into account.

Wagner[2] states that an ionization chamber can be used to assay Tc^{99m} if it in turn has been calibrated against a scintillation crystal method. It is obvious, from our results, that in order to use a crystal scintillation detector, a calibration factor must be determined for any particular geometric arrangement and filtration selected. An ionization chamber dose calibrator may be used to calibrate the crystal system; alternatively, a precalibrated Tc^{99m} source may also be used to standardize the crystal system. Finally, a dose calibrator may be used as the primary assaying device, and we regard this as the method of choice.

SUMMARY

Calibration procedures for Tc^{99m} using a Co^{57} standard and a sodium iodide crystal scintillation detector have been commonly employed. These assays have utilized a variety of detectors, geometries, and attenuators on the assumption that the relative efficiency between the 2 radionuclides is constant.

Our findings demonstrate that the assumption of constant relative efficiencies for all geometries is invalid. Assay of Tc^{99m} should be performed in an ionization chamber or in a crystal detector calibrated for the particular attenuation and geometry selected, using either a calibrated

Tc^{99m} source or an ionization chamber dose calibrator.

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THYROIDAL PERTECHNETATE UPTAKE IN HASHIMOTO'S DISEASE*

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IN A recently concluded statistical analysis of pertechnetate 99m *versus* iodine 131 for the diagnosis of thyroid function, we have suggested that technetium 99m is the most suitable radionuclide for routine clinical thyroid studies.[3] The diagnostic accuracy of thyroidal pertechnetate uptakes is at least as good as that of 24 hour radioiodine uptakes, and technetium 99m possesses advantages over the various radioiodines in thyroid diagnostic procedures in terms of availability, cost, rapidity with which information can be obtained and, above all, low patient radiation dose.

In this report we wish to present the data of all the patients with chronic thyroiditis which had been omitted from the study mentioned above. In patients with chronic thyroiditis there are several metabolic abnormalities which lead to discrepancies in the results of various thyroid function tests, and which had made it, therefore, inadvisable to include these data in the abovementioned statistical analysis.

In an earlier report on the assessment of thyroid function with technetium 99m as pertechnetate, it had been noted that the pertechnetate uptake by the thyroid is elevated above the range of normal in patients with hyperthyroidism and also in patients with thyroiditis.[4] Since then, it has been found that some euthyroid patients who are taking oral contraceptive medication also have elevated pertechnetate uptakes.[3]

The purpose of the present report is to amplify the previously noted observation and to discuss the metabolic abnormality in the patients who had high pertechnetate uptakes but who were neither hyperthyroid nor taking oral contraceptive medication. To complete the picture, all other patients who have had a definite diagnosis of chronic thyroiditis will also be described.

MATERIAL AND METHOD

I. SUBJECTS

Nineteen patients are included in this study. Eight of them have had the diagnosis of Hashimoto's thyroiditis confirmed by surgery or by the finding of high antithyroglobulin antibody titers in the serum. The other 11 patients had elevated thyroidal pertechnetate uptakes but were not hyperthyroid and did not take any medication containing estrogens. Like most of our patients, they had been referred as outpatients for thyroid scannings because of an enlarged and/or nodular thyroid gland. Before the scanning and uptake studies a diagnosis of the functional state of the thyroid was made on the basis of signs and symptoms. The laboratory data shown in Table I were used for confirmation of that diagnosis.

II. PROCEDURES

Thyroid scanning was started about 20–30 minutes after the intravenous administration of ~2.5 millicuries of $Tc^{99m}O_4^-$. Immediately after the patient was scanned, the pertechnetate uptake was determined by scanning a phantom containing 3 per cent of the dose given to the patient. The method for the calculation of the thyroidal pertechnetate uptake has been described before.[4]

Measurements of protein-bound iodine (PBI), thyroxine iodine (T_4-I) and cholesterol were performed on serum obtained at the time of the thyroid scanning. Iodine

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* From the Medical Research Center and Department of Applied Science, Brookhaven National Laboratory, Upton, New York. Research supported by the United States Atomic Energy Commission.

[redacted]

TABLE II

COMPARISON OF THE RESULTS OF THYROID FUNCTION TESTS IN 19 PATIENTS WITH HASHIMOTO'S THYROIDITIS WITH THOSE OBTAINED IN 53 PATIENTS WITH NONTOXIC NODULAR GOITER

	$Tc^{99m}O_4^-$ Uptakes	I^{131-} Uptakes	PBI	T_4-I
Nontoxic nodular goiter (53)*	1.69±0.77†	26.0± 7.2	6.5±1.4	5.0±1.0
Groups I and II (8)	3.98±2.09 (0.001)‡	29.3±13.0 (NS)	3.0±1.2 (0.001)	3.1±0.9 (0.001)
Group III (11)	6.05±2.17 (0.001)	34.5±14.9 (0.01)	4.8±1.1 (0.001)	3.4±0.9 (0.001)
Groups I, II and III combined (19)	5.18±2.37 (0.001)	32.2±14.3 (0.025)	4.1±1.5 (0.001)	3.3±0.9 (0.001)

* No. of patients in group.

† Mean ±SD.

‡ Probability that the value in the test group is identical with the corresponding value in the control group.

NS=Not significant ($p = 0.05$).

10–53 years. Patients 4 and 7 are daughter and mother.

CLINICAL FINDINGS

Of the 19 patients examined, 15 had consulted their referring physician because of the goiter—the other 4 because of symptoms of hypothyroidism. Almost all patients could give a date on which they first noticed the goiter. In most patients the thyroid enlargement was readily noticeable; the enlargement was usually estimated to be 2–4 times normal size. On physical examination the glands were generally firm and diffusely enlarged. The scans, however, not infrequently showed irregular distribution of radioactivity without any definite nodule formation.

LABORATORY FINDINGS

The results of the serum PBI, T_4-I and cholesterol generally confirmed the clinical impression of eu- or hypothyroidism. A PBI–T_4-I difference of greater than 1.5 μg./100 ml. was found in only 2 patients.

Since all our patients had been referred for scanning only, no extensive investigation was done before this pattern of high pertechnetate uptake in eu- or hypothyroid patients was recognized. Tests for thyroid autoantibodies and PBI^{131} values were obtained only recently. In 3 patients neither study was done. Out of 7 patients in Group III, in whom iodide turnover studies were carried out, PBI^{131} values were found to be above the normal range in 4 patients; out of 4 patients with elevated antibody titers, 2 were found to have elevated PBI^{131} values.

The 2 patients who underwent surgery (Group I) had normal antibody titers; 6 patients (Group II) had elevated titers; and 9 patients in Group III with high pertechnetate uptakes had no elevation of antibody titers.

IODIDE AND PERTECHNETATE METABOLISM

Table III shows a comparison of the data obtained. Of the 8 patients in Groups I and II, 4 had normal and 4 had elevated pertechnetate uptakes. Six patients in Groups I and II had normal 24 hour iodide uptakes; however, 1 of them had a high 72 hour PBI^{131} value. Of the remaining 2 patients in Groups I and II, 1 had a low and the other had an elevated iodide uptake. The PBI^{131} value in the patient with the low uptake was, on the other hand, elevated.

The patients in Group III were selected because they had high pertechnetate uptakes. Eight of 11 patients in that group had normal 24 hour iodide uptakes; 4 of the 8 patients had elevated 72 hour PBI^{131} values. Two patients in Group III had ele-

TABLE III

COMPARISON OF THE RESULTS OF PERTECHNETATE UPTAKES WITH THOSE OF IODIDE UPTAKES AND PBI^{131}

	$Tc^{99m}O_4^-$ Uptake	I^{131-} Uptake	PBI^{131}

[redacted]

of iodide metabolism studies in 8 patients.

The finding of an elevated pertechnetate uptake in patients with Hashimoto's thyroiditis is not surprising in view of the known elevation of serum TSH levels in hypothyroid patients.[1,5,9] TSH plays a main role in the regulation of thyroidal iodide transport,[10] and pertechnetate is trapped to an extent almost identical to iodide in the thyroid gland.[2] The results of the radioiodine uptake in Hashimoto's thyroiditis have been more variable, probably because of a decreased iodine pool in the thyroid gland,[11] defective iodide organification,[13] and a subsequent rapid turnover of iodide in the gland.[7] Early 4 hour uptakes have, however, shown good correlation with elevated TSH levels.[9] Our study would suggest that pertechnetate uptakes probably correlate more closely with TSH levels than radioiodide uptakes.

The routine use of the pertechnetate uptake as a test of thyroid function has not been found to be a problem because the time required for calculations is only about 2–3 minutes and the time needed for the additional scanning of the phantom is short. Scannings of the thyroid gland with pertechnetate take less over-all time than those with iodide because a larger dose o the radionuclide can be administered, while the radiation dose absorbed by the thyroid gland is considerably lower.[3]

The importance of our observation is two-fold: (1) it gives another indicator for the confirmation of Hashimoto's thyroiditis, so that the diagnosis can be established without surgery; and (2) it enables identification of individuals who, although euthyroid at the time of the examination, should be followed closely because of the possibility of the development of hypothyroidism.

SUMMARY

Two conditions other than hyperthyroidism will lead to an elevation of the thyroidal pertechnetate uptake.

The first situation is in patients taking medication containing estrogens.

The second situation is in patients with chronic thyroiditis. In patients with this disorder the pertechnetate uptake was consistently higher than expected from over-all thyroid function. In 19 patients the mean pertechnetate uptake was 5.18 ± 2.73 per cent compared to a mean of 1.69 ± 0.77 per cent in 53 patients with nontoxic nodular goiter. This finding is of importance because it aids in the diagnosis of Hashimoto's disease without surgery and it allows identification of individuals who may become hypothyroid in the future.

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RADIOIODINE THERAPY OF THYROTOXICOSIS

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THERE has been considerable discussion concerning the frequency and delayed onset of hypothyroidism after treatment of thyrotoxicosis with radioiodine. Although the use of I^{131} has been regarded as a safe, reliable, and convenient method of treatment, the incidence of hypothyroidism has detracted from its uniform acceptance.[1]

Because of this, and because we have used low dose I^{131} therapy for a number of years to treat thyrotoxicosis, it seemed appropriate to review our series to evaluate its effectiveness and the incidence of hypothyroidism.

In this report the patients were identified according to important characteristics. The significance of these characteristics in predicting a hypothyroid result will be discussed.

METHOD

The charts of all patients treated with radioiodine for thyrotoxicosis from 1963 to September 30, 1969 were reviewed. The patients were classified by the following: (a) sex; (b) age; (c) number of doses; (d) total dose administered; (e) weight of gland, estimated on the basis of physical findings by one of the observers (GLJ); (f) dose delivered, the calculation of which was based upon Quimby's[8] formula:

$$\text{rads} = \frac{90 \times \mu c \text{ (deposited in the gland)*}}{\text{grams (estimated weight of the gland)}};$$

and (g) evaluation of result of therapy.

The patients were referred to the Section of Nuclear Medicine for complete care of thyrotoxicosis. Each patient was followed closely and seen at regular intervals, generally 1, 2, 3, 6 months and yearly after treatment. Each was evaluated, where possible, on the basis of 3 factors (2 of which are quantitative). These 3 factors are:

1. The I^{131} uptake at 4 and 24 hours at least 3 months after the last treatment. An uptake of less than 10 per cent is considered in the hypothyroid range.[7] An uptake greater than 45 per cent is considered to be in the hyperthyroid range.
2. Chemical determination of circulating thyroid substances.† A protein bound iodine determination below 4.0 gamma per cent is considered to be in the hypothyroid range; above 8.0 gamma per cent is in the hyperthyroid range. A T-4 by column chromatography less than 2.9 gamma per cent is in the hypothyroid range; greater than 6.4 gamma per cent is in the hyperthyroid range.
3. A clinical evaluation based upon significant symptoms and physical findings; *e.g.*, weight, temperature, pulse rate, pulse pressure, presence of a fine tremor, eye signs and symptoms, skin and hair texture, sweating, thyroid size, presence of a bruit, deep tendon reflexes, temperature intolerance, fatigue, nervousness, muscle aches, the patient's evaluation of his/her condition, and the referring physician's evaluation.

Where all 3 of these factors (I^{131} uptake, blood chemistry, clinical evaluation) were in the same direction, a final diagnosis was easy. When this was not the case, the final evaluation was based upon the consistent majority.

RESULTS

One hundred and forty-eight patients were treated during this interval. There are 118 females (80 per cent) and 30 males.

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* μc administered × uptake of therapeutic dose

† Majority performed at Bio-Science Laboratories, Van Nuys, California.

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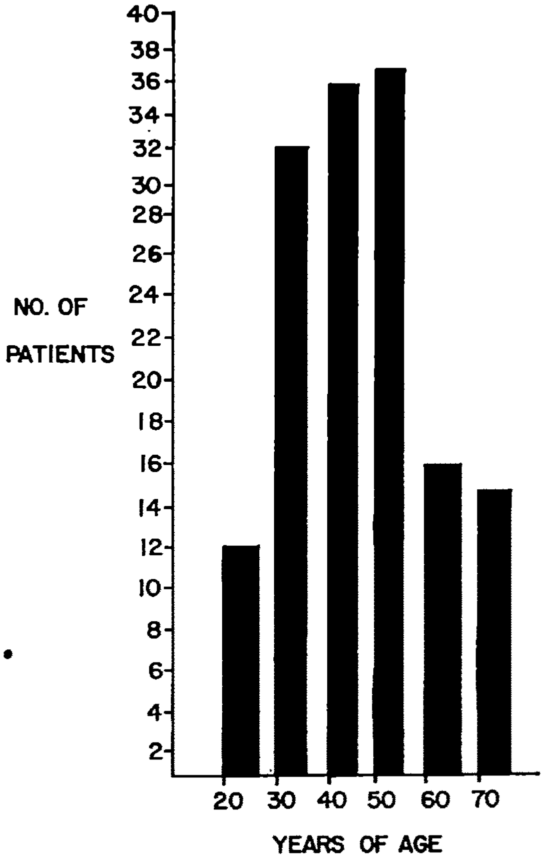

Fig. 1. Ages of 148 patients treated with radioiodine for thyrotoxicosis.

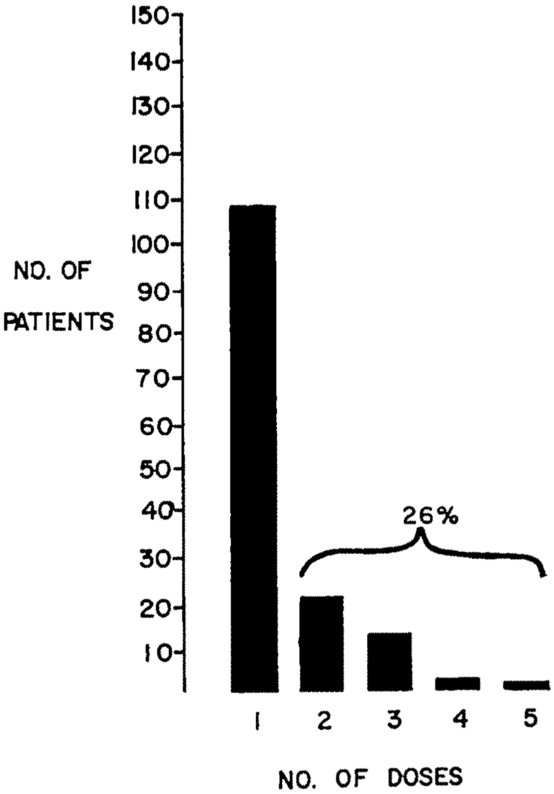

Fig. 2. Number of treatment doses of radioiodine administered to 148 patients.

The hypothyroid patients were tabulated according to the various subsets (Table III).

From Table III the following significant observations are noted:

1. No patient received more than 7 mc.
2. The majority (13 of 15 = 87 per cent) had 1 treatment.
3. The estimated weight of thyroid glands was 65 gm. or less.
4. The delivered dose exceeded 3,500 rads in 14 of the 15 patients.

Table II

RESULTS OF RADIOIODINE THERAPY FOR THYROTOXICOSIS

	I^{131} Uptake		PBI or T_4		Clinical		Final Evaluation	
	No.	Per Cent	No.	Per Cent	No.	Per Cent	No.	Per Cent
Hypothyroid Range	19	15	13	11	14	11	15	11
Euthyroid Range	99	79	75	65	113	86	112	85
Hyperthyroid Range	7	6	28	14	4	3	4	3
No Follow-up Data	23		32		17		17	

Table III

RADIOIODINE INDUCED HYPOTHYROIDISM

Patient No.	Sex	Age	No. of Doses	Total Dose (mc)	Weight of Gland (gm.)	Dose Delivered (rads)	Onset of Hypothyroidism (yr.)	Treatment Uptake (per cent)
85	F	38	1	2.5	35	3,800	4	59
117	F	50	1	4.0	25	6,900	¼	48
231	F	63	2	3.0 4.0	20 20	16,700	½	53 53
370	F	51	1	4.0	30	6,600	¼	55
429	F	47	1	4.0	45	4,700	¼	59
561	M	46	2	4.0 1.55	50 20	7,100	¼	70 30
756	F	53	1	4.0	30	9,400	2	78

[redacted]

measures of hypothyroidism would have placed 13 of our subjects in the hypothyroid category. If one examines the hyperthyroid group as identified by I^{131} studies, there would be 7 in this category. The chemical evaluation would have listed 28 as hyperthyroid. It thus seems that strict adherence to either the uptake or chemical determination would introduce a distortion. In fact, then, we believe that this 3 part evaluation has more safeguards than bias. It also reflects our opinion of the significance of clinical judgment.

It should be noted (Fig. 3) that the median total dose of I^{131} administered is less than 4 mc. When compared with other reports in the literature[3,6,9] this is found to be quite low. Similarly, the incidence of hypothyroidism is lower than that frequently reported.[3] A recently published exception is the prospective study of Smith and Wilson.[10] They report 8 per cent of patients receiving a conventional dose of I^{131} (*i.e.*, 140 μc/gm. of thyroid, delivering about 7,000 rads, 3–7 mc range) became hypothyroid in the first year. Twenty-nine per cent became hypothyroid in 5 years. For patients receiving a half-dose (70 μc/gm. of thyroid, delivering about 3,500 rads, 1.2–2.4 mc range) the hypothyroid result is 4 per cent at the end of 1 year and 7 per cent at 5 years. Our results fall between the conventional and half-dose group of Smith and Wilson as seen in Figure 5. Their conventional and half-dose curve plotted in a similar manner is superimposed. The derivation of this curve is shown in Table 1.

The annual increment of hypothyroidism seen with conventional doses of radioiodine is not seen in the series reported here.

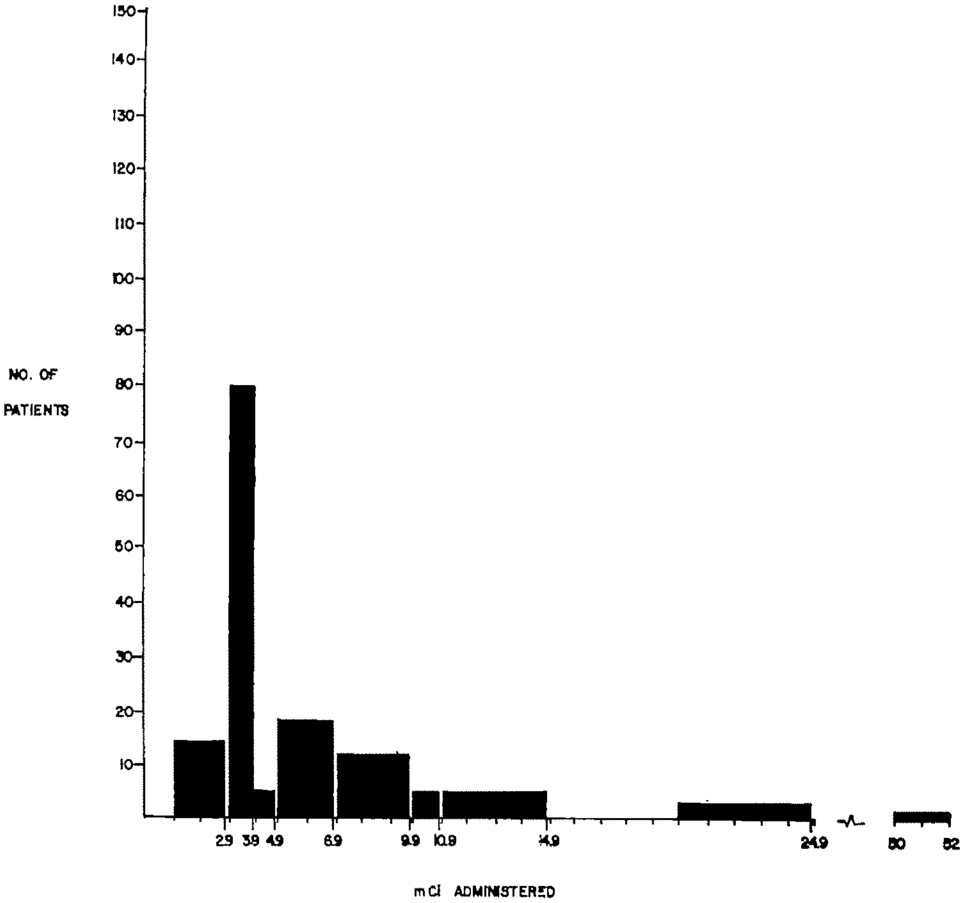

Fig. 3. Total treatment dose of radioiodine (in millicuries) administered to 148 patients.

NUMBER
OF
PATIENTS

INCIDENCE OF HYPOTHYROIDISM

40

•—• Smith & Wilson, BMJ

[redacted]

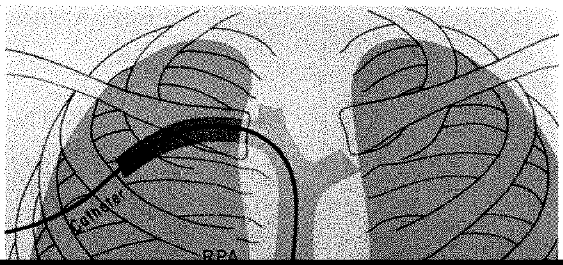

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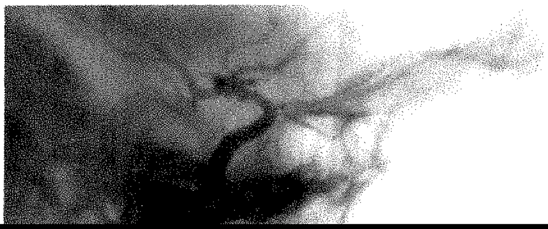
FIG. 3. Hazards of intra-arterial irradiation of brain tumors. (*A*) Y^{90} microspheres (arrows) lodged in the vessels of the retina and (*B*) in the ~~vessels of the sclera (arrow). This is avoided by pressure on the globe~~ of the eye during the injection of the microspheres.



macula by ophthalmoscopy. Injection into the ophthalmic artery is now avoided by pressure on the globe of the eye exceeding the systolic blood pressure during the minute or two it takes to inject the suspension of microspheres.

What about the tolerance of the brain to

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into one pulmonary artery via catheter (Fig. 1, *A* and *B*), while the other unirra-

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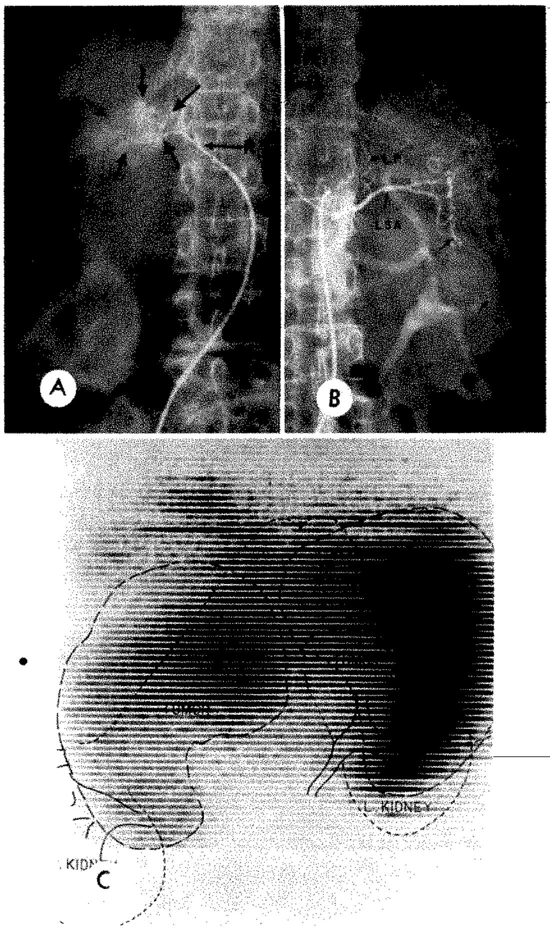

FIG. 8. Metastatic tumors in adrenals from carcinoma of the lung. (*A* and *B*) Angiograms show contrast material within the tumors (arrows). (*C*) After injection of microspheres the same distribution is demonstrated in the Bremsstrahlung scan. R = right inferior adrenal artery; LP = left phrenic artery; LSA = left superior adrenal artery.

patient selection at present is limited to hopeless cases. We have seen several cases where a liver enlarged with metastases showed definite shrinkage following treatment. Intra-arterial irradiation is a technique of treating a tumor simply and safely, without the systemic reaction associated with external irradiation. In addition, some tumors can be treated to a dose level not possible by external means because of the radiosensitivity of surrounding structures. This is especially true in the abdomen where the tolerance of the small bowel is a limiting factor.

The most satisfying results of this form of treatment have been in patients with the carcinoid syndrome resulting from metastatic functioning tumor of the liver.[12,13] Almost complete subsidence of symptoms followed injection of the radioactive microspheres into the hepatic artery. A decrease in the size of the liver metastases has been shown in some cases. Remissions may last for a year. We have retreated 4 patients, at yearly intervals, without any significant signs of impairment of liver function. One patient was treated 4 times. It is hoped that it will be possible, by this method, to delay the development of the cardiac lesions associated with carcinoid syndrome.

From a technical standpoint, catheterization of a specific vessel presents no problem. The percutaneous femoral approach is usually adequate, but, when there is distor-

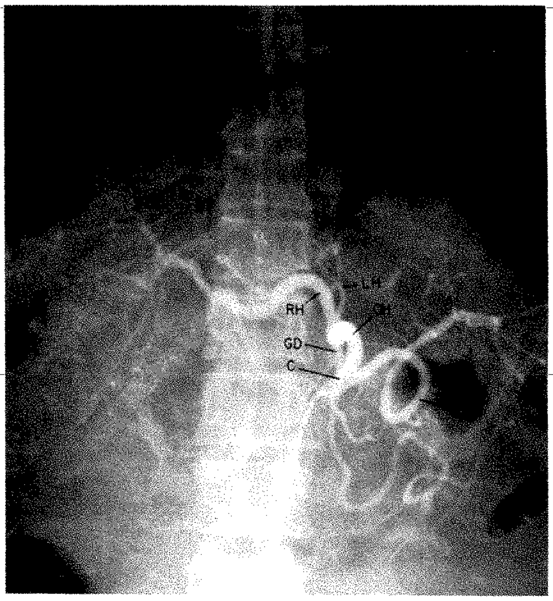

FIG. 9. Selective percutaneous celiac arteriogram shows marked tortuosity of the vessels in an 80 year old woman with carcinoid tumor of the right lobe of the liver. Attempts to catheterize the right hepatic artery (RH) failed. No radioactive microspheres were injected. LH = left hepatic artery; CH = common hepatic artery; GD = gastroduodenal artery; S = splenic artery.

tion of the celiac axis, an approach via the Norman Simon, M.D.

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SURFACE APPLICATORS AND INTERSTITIAL IMPLANTATIONS WITH RADIOACTIVE IRIDIUM 192*

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RECOGNIZING the risk of radon gas escape and a high dose to personnel, only a few centers are still using radon seeds as the radioactive material for surface applicators and interstitial implantations. In 1957, Henschke[2] introduced the technique for permanent implantation of iridium 192 (Ir^{192}) seeds. A special apparatus with a depth gauge and needles (21 gauge) were employed for the introduction of the required number of seeds (0.35 mm. in diameter) at precalculated depths in the tissue. Meurk *et al.*[4] have noted that the Paterson and Parker data are valid at several distances for the planar implants. By measurement in a water phantom they found that beyond 6 cm. the attenuation curves for gamma rays of Ir^{192} deviated appreciably from the inverse square law. The suitability and advantage of the physical properties of Ir^{192} for interstitial implantations, as compared to either radon 222 or gold 198 seeds, have been discussed by Henschke.[3]

In this paper our experience with iridium 192 for surface applicators is reported and the superiority of Ir^{192} over Au^{198} for surface applicators is described. Also, the distribution for the arrangement of iridium 192 seeds of various shapes for interstitial implantation is tabulated in a convenient form.

MATERIAL AND METHOD

The radioactive iridium 192 seeds used in our department are supplied by BARC (The Bhabha Atomic Research Center, India). Each consignment consists of 100 seeds of approximately 300 microcuries each, lasting on the average for a period of 2 to 3 months. The activity per seed is measured by a re-entrant ionization chamber using a radium standard. Occasionally a maximum difference of 15 per cent was observed between the measured dose and the stated value.

SURFACE APPLICATORS

Thin transparent perspex or stellon sheet of a thickness 2 to 3 mm. was found more suitable for mounting the Ir^{192} seeds than either foam rubber or felt (not elastoplast). The considerable reduction in weight of the perspex with respect to either the dental compound or the plaster of Paris is an advantage. Foam rubber has the disadvantage of stretching during application on the patient resulting in an increase in the active area with a consequent fall in dose rate. The felt that was locally available was found to be too thick. This resulted in an increase in the percentage depth dose at greater depths, which was undesirable in a few cases.

Dosimetry. The dosimetry for the Ir^{192} surface applicator is based on the equivalent dose in mc hr. per initial mc of the isotope (Table I). The calculation is made using a value of 4.8 for the K-factor of Ir^{192} instead of the value of 5.5 used by Henschke.[3] It is seen that a 24 hr. decay of 1 millicurie Ir^{192} gives an equivalent dose of about 24 mc hr. per initial mc, which compares favorably with the corresponding values of 21.96 and 21.17 for Rn^{222} and Au^{198}, respectively.

Table II outlines an irradiation schedule for a given case, where a tumor dose of 5,000 rads is planned to be delivered in 6 applications of 12 hours each in 6 days. The total activity required to deliver a dose of 840 rads in 12 hours per application, is calculated to be 8.26, 18.5 and 5.93 millicuries for Ir^{192}, Au^{198} and Rn^{222}, respectively. Seeds

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[redacted]

Table IIIA

DISTRIBUTION OF IRIDIUM 192 SEEDS OF INITIAL STRENGTH OF 225 MICROCURIES TO GIVE A DOSE OF 18,000 RADS AT ½ CM. FROM THE PLANE OF PERMANENT IMPLANT

CIRCLES

Diameter of circle (cm.)	1	1½	2	2½	3	3½	4	4½	5	6	7	8	9	10
Area in cm.^{2}	.78	1.8	3.1	4.9	7.1	9.6	12.6	15.9	19.6	28.3	38.5	50.3	63.6	78.6
Total number of seeds	3.1	4.7	6.3	8.2	9.9	11.8	13.9	16.2	18.5	24.2	30.2	36.4	42.9	49.8
Seeds in periphery	3	4	4	5	7	8	9	10	10	13	15	18	22	25
Seeds over area	—	1	2	3	3	4	5	6	8	11	15	18	21	25

The relative proportion of seeds around the periphery and over the area is applicable for loading surface applicators of corresponding geometry, with proper activity per seed chosen (*e.g.*, 1 to 1.5 mc per seed).

of activity of 1 mc of Ir ^{192}, 3–4 mc of Au^{198} and 0.5 mc of Rn^{222} are suitable for loading the surface applicator. These may be arranged as stipulated in Table IIIA-D, depending on the shape of the lesion.

The advantages of iridium over either radon or gold are as follows:

(a) With Ir^{192} the increase in treatment time for subsequent days to deliver the same dose (840 rads) to the area of interest is only about 1 per cent on the second day of application, rising to a maximum of about 6 per cent on the sixth day, while in the case of Au^{198} even on the second day it is as high as 30 per cent, rising to impracticable values on subsequent days. Therefore, Ir^{192} is a better radioactive material for such intermittent applications over an extended period.

(b) The maximum dose at 50 cm. from the applicator is estimated to be 13.7 mr/hr. for Ir^{192}, 17 mr/hr. for Au^{198}, and 19.7/hr. for Rn^{222}.

(c) With Au^{198}, the dose rate during a single application (12 hr.) would fall from 70 rads/hr. to 60 rads/hr. towards the end of the treatment each day, while the dose rate fall with Ir^{192} is negligible.

(d) The required beta shielding is less with Ir^{192} than with Au^{198}.

(e) Compared to radon seeds, the risk of

Table IIIB

DISTRIBUTION OF IRIDIUM 192 SEEDS OF INITIAL STRENGTH OF 225 MICROCURIES TO GIVE A DOSE OF 18,000 RADS AT ½ CM. FROM THE PLANE OF PERMANENT IMPLANT

SQUARES

Side of square (cm.)	2½	3	3½	4	4½	5	6	7	8	9	10
Area in cm.^{2}	6¼	9	12¼	16	20¼	25	36	49	64	81	100
Total number of seeds	9.3	11.4	13.6	16.2	19.1	22.1	28.8	35.8	43.1	51	59.5
Number of lines	3	4	4	4	5	5	5	7	7	8	8
Distance between lines (cm.)	1.25	1	1.2	1.3	1.1	1.25	1.5	1.2	1.3	1.3	1.4
Number of seeds in each line			4			5	6	6	7	8	8
	3	3	3	4	4	4	5	5×5	5×6	6×6	6×7
			3			4	5	6	7	8	8
			4			4	5				
						5	6				

Treat squares 1×1 and 1½×1½ as circles

The relative proportion of seeds around the periphery and over the area is applicable for loading surface applicators of corresponding geometry, with proper activity per seed chosen (*e.g.*, 1 to 1.5 mc per seed).

Table IIIC

DISTRIBUTION OF IRIDIUM 192 SEEDS OF INITIAL STRENGTH OF 225 MICROCURIES TO GIVE A DOSE OF 18,000 RADS AT ½ CM. FROM THE PLANE OF PERMANENT IMPLANT

[redacted]

with stylets of suitable length (15.5 and 24 cm., respectively). The average outer diameters of the cannula and the stylet are 1.25 mm. and 0.75 mm., respectively. A spherical head of about 4.0 mm. (dia.) on each cannula provides a good grip and control in implanting the seed at the required site in the tissue. The cannula ends in a sharp point with a beveled edge of about 2 mm. length in the proximal end. In the case of a single plane implant, 1 cannula is used for each seed. For double plane or volume implants, however, the same cannula may be used for 2 or 3 seeds along a given plane by withdrawing the cannula through known distances.

An approximate calculation of the required dose in the case of Ir^{192}, applying the dose-time relationship

$$\frac{D_1}{D_2}=\left(\frac{T_1}{T_2}\right)^{0.26} \quad \text{of Ellis,}^{1}$$

gave a value of 12,000 rads for producing the same effect in 107 days as against 6,000 rads in 7 days by radium. Since the above relationship was primarily intended for use with radium only, the tumor dose prescribed here was increased to 18,000 rads. The choice of the higher dose was supported by the clinical results in producing the desired reaction in the region of interest.

For the distribution of Ir^{192} seeds, the data used for Au^{198}, at the Hammersmith Hospital, London, were modified to suit the activity per seed of Ir^{192} and the dose of 18,000 rads prescribed. These data are shown in a convenient form in Table III A-D. The total number of seeds required for a given case arrived at by following the data in Table III and that arrived at by calculation in the usual way agree within 5 per cent. The distribution rules stipulated are also applicable for surface applicators, but with higher activity per seed (*e.g.*, 1 mc for Ir^{192}).

SUMMARY

1. Radioactive iridium 192 is a better material than Au^{198} for surface applicators using intermittent applications over a period of 2–6 days.

2. The activity per applicator is about 50 per cent as compared to that with Au^{198} for a given case.

3. The dose to personnel is as low as 13 mr/hr. at 50 cm. from the surface of the applicator containing about 8 mc Ir^{192}. This dose could be further reduced to negligible proportions by a lead shield of 2–3 mm. In the case of radium the dose would be much higher and a larger thickness of lead sheet would be required for a comparable reduction in dose at 50 cm.

4. While loading the applicator with Ir^{192}, the protection required is reduced nearly to one-third of that required for radium.

5. The distribution rules given in Table IIIA-D are more convenient for use by the clinician in terms of effort and time.

6. For the interstitial application of Ir^{192}, special apparatus, like the gold grain gun, is not essential.

7. Hospitalization of the patient can be minimized.

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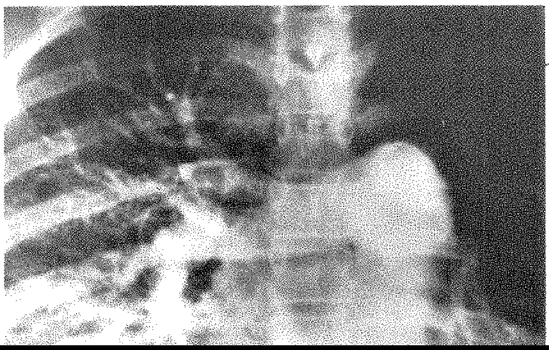

portant cardiac arrhythmias and, consequently, does not require cardiac monitoring and defibrillation equipment. There is no risk of ventricular perforation, and catheter manipulation time is minimal.

The scannings were performed with I^{131} tagged macroaggregated human serum albumin injected intravenously in the prone position. Patients were scanned in the supine position, using a rectilinear scanner with 2 opposing detectors. The signals from

[redacted]

Table I

37 positive scans

Emboli	17
Emphysema (localized or generalized)	7
Effusions (isolated pleural)	5
Infiltrate (pneumonia)	4
Atelectasis (discoid or lobar)	2
Asthma	1
Cardiac Failure (patchy uptake)	1

had positive arteriograms, although in theory such would be possible, especially in the left lower lobe where the heart produces a normal scan defect, which may adversely affect the interpretation.

CONCLUSIONS

1. Pulmonary scanning is an accurate, but nonspecific, method of evaluating pulmonary perfusion as shown by a high degree of correlation with pulmonary arteriography and plain film findings (94 per cent over-all agreement). Where the scan was negative, the pulmonary arteriogram was also negative in all cases. However, in 1 case the scan was positive on the right (middle lobe) and normal on the left, while the arteriogram showed a left lower lobe embolus. Apparently this was not identified on the scan because of its retrocardiac location. It would seem prudent to conclude that in the evaluation of pulmonary embolus the presence of a negative scan makes the likelihood of a positive arteriogram very slight, but is not completely exclusive with reference to the retrocardiac area.

2. Approximately one-half (54 per cent) of the scan abnormalities found were associated with disease processes other than embolism in spite of the clinical suspicion of embolism which led to the performance of the examinations. This finding is of clinical importance in terms of patient treatment and suggests that positive scans should be followed up by arteriography for complete evaluation.

3. A simple method of pulmonary arteriography and scanning is described which can be performed safely and rapidly in virtually any X-Ray Department. We feel that it is particularly suited to the patient in acute distress due to the presence of recent pulmonary embolization.

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THE VALUE OF POSTOPERATIVE INTRAPERITONEAL INJECTION OF RADIOCOLLOIDS IN EARLY CANCER OF THE OVARY*

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THE intraperitoneal injection of radiocolloids was introduced in the management of cancer of the ovary 25 years ago. The rationale for this method was to pre-

[redacted]

tive pelvic irradiation does not contribute to the survival of patients in whom complete resection of the ovarian cancer was possible. Total abdominal irradiation, how-

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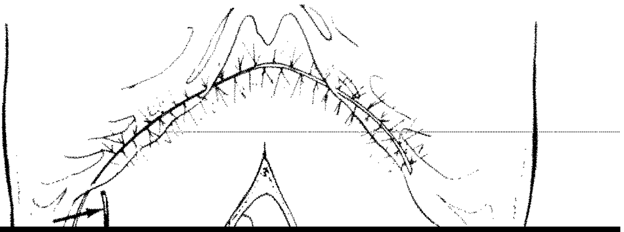
attributable to the instillation of radioactive phosphorus.

TECHNIQUE OF INSTILLATION OF RADIOACTIVE COLLOID

The injection of radioactive phosphorus is done soon after surgery through 2 nylon



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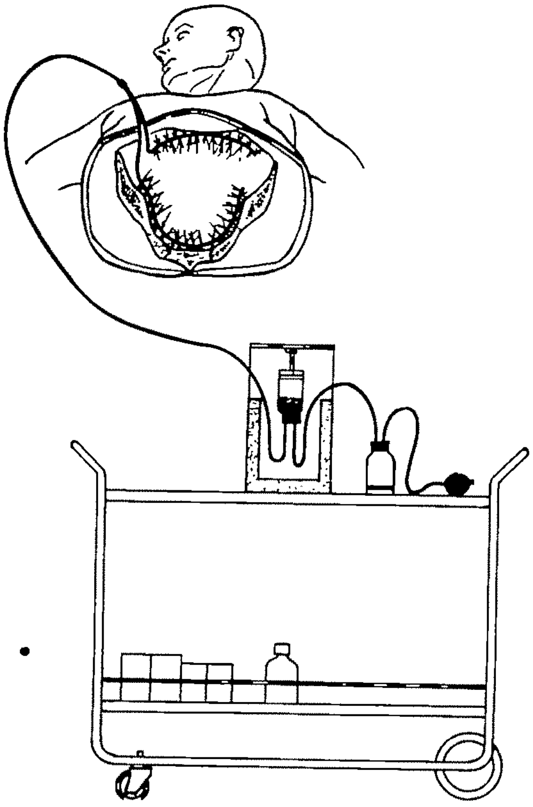

FIG. 2. Injection of the colloidal radioactive phosphorus under pressure.

radioactive phosphorus, 2 patients died of cancer at 13 and 28 months after surgery.

If one eliminates the 4 patients who died without disease and the 1 patient who died postoperatively, a total of 26 patients is left in the group with surgery only. At the end of 5 years, 20/26 patients (77 per cent) in the surgical group and 24/26 patients (92 per cent) in the group with radioactive phosphorus were alive without evidence of ovarian cancer. This difference is of borderline statistical validity, since approximately 50 patients in each group would be required to detect a 15 per cent difference (from 77 to 92 per cent with a p 0.05).

In Table IV, a more detailed analysis of the 5 year survivors is presented in an effort to weigh the influence on survival of various other factors, such as pelvic irradiation, omentectomy, histology, and extent of disease. Statistical analysis of these results (using the Chi-square test and Fisher's exact probability with Tocher's modification) shows no significant difference between the 2 groups, however, because of the small number of patients involved.

Figure 4 shows the percentage of recurrences in the 2 groups of patients; *i.e.*, the patients with surgery only, and the patients with surgery and postoperative intraperitoneal injection of radioactive phosphorus. Seven patients (22.5 per cent) in the first group, and 3 patients (11.5 per cent) in the second group developed local recurrences after their treatment at Memorial Hospital. In only 1 patient in each group was the recurrent ovarian cancer eventually controlled. The first was a 57 year old housewife with a granulosa cell carcinoma of the right ovary treated by BSO and P^{32}; a hysterectomy was not performed because of her poor general condition. Five and one-half years later an abdominal recurrence was noted and the patient underwent a TAH and omentectomy at this time. She is at present alive and well, 12 years after the initial treatment and 6½ years after the treatment for recurrence. The second patient was a 60 year old housewife who also had a granulosa cell carcinoma of the right ovary. A BSO was performed and postoperative pelvic irradiation (3,000 rads) was given. Nineteen months later, an abdominal recurrence was noted. The patient underwent a TAH and omentectomy followed immediately postoperatively by the intraperitoneal in-

TABLE IV

STAGE I—CANCER OF THE OVARY
5 YEAR SURVIVAL
1956–1965

	Surgery	Surgery and P^{32}
Pelvic irradiation		
yes	6/10	8/9
no	14/21	16/17
Omentectomy		
yes	4/5	14/16
no	16/26	10/10
Papillary adenocarcinoma	12/23	18/19
Other	8/8	6/7
Unilateral involvement	16/22	15/16
Bilateral and/or ascites	4/9	9/10

jection of 10 mc of P^{32}. She has been on cytoxan since then and is at present alive without evidence of cancer 8 years after the original treatment and almost 6½ years after treatment for the recurrence.

The remaining 8 patients died of uncontrollable cancer following the recurrence of their disease. Seven of these patients had papillary carcinoma, and 1, a pseudomucinous adenocarcinoma. Thus, the important point in this report is the significant prolongation of a symptom-free period in the group receiving P^{32}. Even though survival percentages and length of survival might be identical in the 2 groups, the symptom-free interval has been extended appreciably. This achievement more than vindicates the use of P^{32}.

Table V

STAGE I—CANCER OF THE OVARY
CAUSE OF DEATH
1956–1965

	Surgery	Surgery and P^{32}
Local recurrence	2	2
Local recurrence and distant metastases	4	0
Second cancer	2	0
Other	3	0
All causes	11	2

death, which are listed in Table v. Two patients in each group died of local (intra-abdominal) recurrence. Four patients in

[redacted]

with surgery only developed late complications; 1 peritoneal adhesions, 1 intestinal obstruction, and 1 a severe fatal glomerulonephritis. One patient (4 per cent) in the group with radioactive phosphorus developed severe hydronephrosis and cystitis. The first of these 4 patients had surgery only; the second patient received pelvic irradiation postoperatively; the last 2 patients received total abdominal irradiation postoperatively (3,000 and 4,200 rads, respectively).

SUMMARY

1. From April 1956 to September 1965, 57 patients with Stage I cancer of the ovary (as defined by the International Federation of Gynecologists) were treated at Memorial Hospital. The over-all 5 year survival rate of these patients is 78 per cent (44/57).

2. Radioactive colloidal phosphorus (P^{32}) was used in 26 patients (46 per cent of all patients). The 5 year survival rate for the patients with and without P^{32} is 92 per cent (24/26) and 64 per cent (20/31), respectively.

3. There is no evidence of any major adverse effect, either in the postoperative period or at a later date, attributable to the instillation of P^{32}.

4. Because of the small number of patients, no definite conclusions can be formed at this time regarding the value of omentectomy. A controlled prospective study, therefore, appears desirable in order to investigate this question.

5. It appears that the postoperative instillation of P^{32} decreases the incidence of local (intra-abdominal) recurrences, but owing to the small number of patients, no definite conclusions can be drawn.

6. A controlled prospective study on a nation-wide basis is advisable in order to determine the value of radiocolloids in early cancer of the ovary, since only a small number of patients of this type are seen at any one institution. On the basis of our preliminary results, we at Memorial Hospital are presently using colloidal P^{32} immediately postoperatively in all patients with Stage I cancer of the ovary; in Stage II, the patients are randomized following surgery for radioactive phosphorus plus external pelvic irradiation *versus* chemotherapy alone.

The main argument for the use of colloidal phosphorus postoperatively is the simplicity of administration and the absence of severe side reactions and late effects, which are associated with the use of total abdominal irradiation.

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SECOND LOOK SURGERY AFTER RADIATION THERAPY FOR ADVANCED STAGES OF CANCER OF THE OVARY*

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THE results of treatment of advanced carcinoma of the ovary are poor.[1,2,5,8,9,11] The disease that has spread beyond the pelvis is almost universally fatal. Nevertheless, treatment must be given in an attempt to arrest the disease and prolong comfortable survival. Combinations of various treatment methods have been employed;[2,5–7,11,14] however, it remains unclear which are most effective.

The role and scope of surgery in primary treatment of incompletely resectable, advanced ovarian cancer is still disputed. Many authorities advocate removal of as much disease as is feasible.[4,5,11] Some even advise radical resection of involved pelvic and abdominal viscera.[1]

Others believe that incomplete surgical excision may actually disseminate disease by opening lymph and blood channels, and spread cancer cells over the peritoneum.[9] Kottmeier states that recurrence follows rapidly after forcible excision of fixed masses.[6] Irradiation and chemotherapy, prior to excisional surgery, have been recommended instead.

In 1960, we began a program of combined treatment for advanced ovarian carcinomas. It consisted of exploratory laparotomy, biopsy, irradiation and concurrent chemotherapy, followed by second look surgery. The aim of preoperative treatment was to destroy tumor masses, or eliminate their fixation; and thereby aid definitive surgery and prevent iatrogenic dissemination.

The purpose of this paper is to present 17 cases who received this combined treatment. All were followed for a minimum of 3 years.

TREATMENT POLICY

Exploratory Laparotomy. Laparotomy was performed in all cases of suspected carcinoma of the ovary. Advanced disease, incompletely resectable, with fixed masses or peritoneal spread, was left undisturbed except for biopsy.

Irradiation. A course of 3,000 rads abdominopelvic irradiation was given after wound healing, supplemented by 1,000 rads to the pelvis if tumor fixation persisted.

Chemotherapy. ThioTEPA was administered intramuscularly concurrent with the irradiation. A total dose of 1.5 mg./kg. of body weight was aimed at. In cases in which hematopoietic depression developed, chemotherapy was interrupted temporarily without discontinuance of radiotherapy.

Second Look Surgery. Re-exploration was undertaken 4 to 8 weeks after completion of irradiation and at least 3 weeks after chemotherapy if the clinical response to treatment was satisfactory. No surgery was performed if the patients were unimproved, or had progression of disease. Definitive surgical treatment consisted of total abdominal hysterectomy (TAH) and bilateral salpingo-oophorectomy (BSO), plus omentectomy.

Biopsy, intestinal palliative bypass, or supracervical type hysterectomy was done in cases with incompletely resectable disease.

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CLINICAL MATERIAL

During the period reviewed, 1960–1966, 37 patients with advanced carcinoma of the ovary received irradiation and chemotherapy after laparotomy. Seventeen of these had second look surgery and form the basis of this report.

The other 20 patients were not re-explored because of: (1) incomplete treatment (5 cases); (2) poor response or distant spread (8 cases); (3) refusal to undergo reoperation (5 cases); and (4) medical contraindications (2 cases). These last 7 patients had good clinical response to irradiation and chemotherapy.

Age. Patients with second look surgery varied in age from 20 to 71 years. The age range was as follows: 20–29 years, 1; 30–39 years, 4; 40–49 years, 4; 50–59 years, 5; 60–69 years, 2; and 70–79 years, 1.

Clinical Staging. Tumors were staged retrospectively according to the recommendations of the International Federation of Gynecology and Obstetrics (adopted in 1964).[8] The findings were: Stage IIB—2; Stage III—14; and Stage IVA—1.

Ten of 17 patients had gross ascites, 2 had liver metastases; 4 had pleural effusion, 1 of whom had proven metastases to the pleura.

Histologic Classification. The tumors were of the following types: papillary serous cystadenocarcinoma—12; pseudomucinous adenocarcinoma—2; granulosa cell carcinoma—2; and undifferentiated carcinoma—1.

Surgical Procedure at Exploratory Laparotomy. Biopsy only was performed in 14 patients. Salpingo-oophorectomy was done in 3 cases; unilateral in 2, and bilateral in 1.

Radiotherapy. Irradiation was administered with a 250 kv. constant potential unit, HVL. 3 mm. Cu. Opposing abdominopelvic ports were used, extending from the xiphisternum to the upper or mid-symphysis. Usual field sizes were 22–24 cm. by 30–35 cm. Usually, one kidney was blocked during posterior irradiation.

A midplane tumor dose of 3,000 rads in 6 to 8 weeks was given in most cases. The dose was slightly reduced or the treatment time prolonged in a few patients in poor condition, or with severe radiation reactions. An additional 1,000 rads was given in 1 to 2 weeks with the cobalt 60 unit to the pelvis if pelvic fixation remained.

Chemotherapy. ThioTEPA was administered 3 times per week: 2.5 mg. doses concurrent with radiotherapy, and 5 mg. doses thereafter. Chemotherapy was interrupted in a few patients with leukopenia below 2,000 or platelet depression below 80,000, but was terminated before completion in only 2. Total doses ranged from 32 to 125 mg., and averaged 77 mg.

Second Look Surgery. Irradiation produced significant regression of tumor in all but 3 cases. The masses became smaller and more mobile; the peritoneal implants disappeared. Ascites was completely controlled in 9 of 10 patients with fluid at initial laparotomy. In 9 patients gross total removal of tumor was achieved: 3 with complete regression, and 6 with sufficient regression to permit total excision. Five had incomplete removal. In 3 other patients no resection was attempted because the residual disease was extensive and the fixed masses persisted (Table 1).

The surgical procedures performed were: abdominal hysterectomy, salpingo-oophorectomy and omentectomy in 13, 4 of whom had a supracervical-type hysterectomy; bilateral salpingo-oophorectomy in 1 with previous hysterectomy; and biopsy only, or palliative intestinal bypass in 3.

Histologic studies showed residual microscopic disease in the surgical specimens or biopsies in all 17 cases.

RESULTS

The results of treatment are given in Figure 1. Ten of the 17 patients were dead in less than 2 years of pelvic and abdominal recurrences. Seven survived over 3 years (41 per cent), 4 with repeated courses of chemotherapy and/or irradiation for recurrence.

The average survival from the time of diagnosis of the whole group was 29

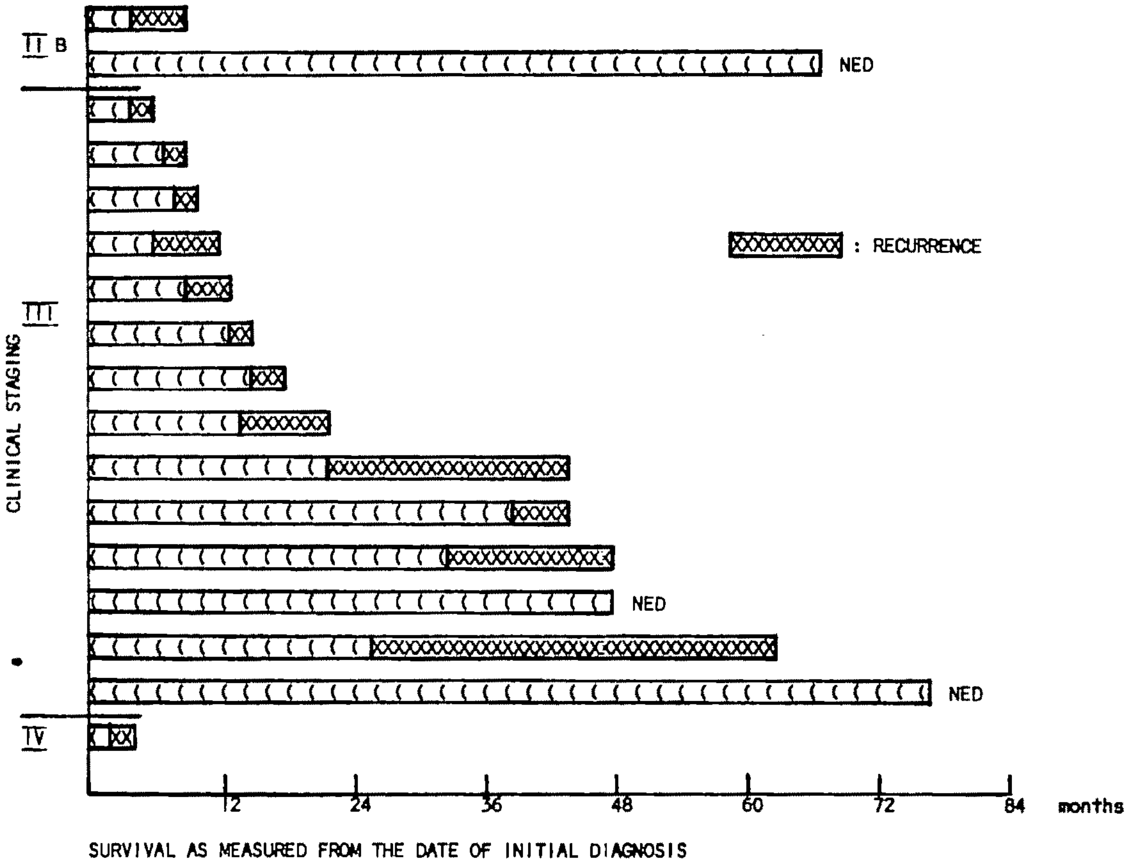

FIG. 1. Graph shows duration of survival and recurrence-free interval in months from the time of initial diagnosis. Three patients are still alive and well. NED = no evidence of disease.

main to be determined. No definite conclusions can be drawn from this small study; however, kilovoltage irradiation and small doses of thioTEPA achieved sufficient tumor destruction to permit gross total removal in the majority of cases re-explored.

More intensive preoperative treatment with megavoltage techniques[3] and the newer chemotherapeutic agents[14] should prove more effective and merit clinical trial.

CONCLUSION AND SUMMARY

1. A series of 17 patients with advanced ovarian carcinoma who had exploratory laparotomy, 3,000 rads abdominopelvic irradiation, thioTEPA and second look surgery is reported.

2. Tumor regression was complete or sufficient to permit gross total removal of the tumor in 9 of 17 cases. Microscopic disease was present in all cases, however.

3. A 3 year survival of 41 per cent was achieved. The average survival of all cases was 29 months; 20 months free of recurrence. Three of the 17 cases remain free of disease from 48 to 77 months.

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SPATIAL STATISTICS OF A GAMMA CAMERA

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NUCLEAR Medicine is a rapidly growing, primarily patient-oriented spe-

tainer. This is placed at a uniform distance from the detector face (Fig. 1). Some stud-

[redacted]

numbers). Normalizing the histogram in the abscissa requires converting all the data to a *normalized* form by first subtracting the average value from every data element and then dividing the result by the standard deviation. When this is done, the data values on the X axis will always be centered about zero and extend for, typically, 3 to 6 units in each direction from zero.

The histograms used in this computer program are normalized in both the X and

is also the over-all frame maximum or minimum value, an X (maximum) or an N (minimum) is indicated. Thus, the co-ordinates of the maximum and minimum values can readily be determined by scanning the row and column sections. With the exception of the X and N indications, the frame, row, and column sections are identical in format and meaning.

The next 2 sets of numbers printed are the range and the median value for the set of

[redacted]

upper confidence limits, and those to the left, the lower confidence limits. We have preferred to use 95 per cent limits, suggesting that the true average value or true variance could be found within this range with 95 per cent confidence.

The next 2 numbers produced in the program are skew and kurtosis (Fig. 2). It can be shown that the average, variance, skew, and kurtosis are related since each arises in a certain power series expansion in a characteristic function that represents the statistical distribution at hand. It is known that for a normal distribution the skew is 0, and kurtosis, 3. A statistical test which will designate the significance of the departure of skew and kurtosis parameters from certain desired values is not known. However, these parameters give a qualitative measure of the "fit" to any selected distribution. Should the data be shown to not be normal, these 4 parameters may be used as a guide in determining a suitable distribution for further study.

The next 2 columns, std-dev and 95 per cent L+95 per cent H, must also be considered as a set (Fig. 2). Here, the standard deviations of the data set with an associated confidence interval are defined. These numbers represent the square roots of the values given for the variance.

The next column of data is the coefficient of variation (Fig. 2). This number is computed by dividing the standard deviation by the average.

The next to the last column contains Chi square statistics computed for a normality test of the data set (Fig. 2). It is here that the cell center and cell width parameters previously discussed impact as the statistic is determined from a histogram prepared from the raw data. This is also true for the next column of numbers represented as K-S (Kolmogorov-Smirnov statistic). The degrees of freedom depend upon the cell width and are shown in column, NU.

The final column of the number table is the Kolmogorov-Smirnov (K-S) statistic (Fig. 2). This number is computed from a normalized cumulative histogram by finding the maximum difference at any cell edge between the sample histogram and the true normal histogram. The cumulative histogram differs from the histogram described above in that the total number of elements less than the chosen cell edge are used when determining frequency rather than using the number of elements between cell edges.

For both the Chi square and the Kolmogorov tests, the computed statistic is compared against a table of previously tabulated values to determine whether the data can be considered normal.[4,5] The confidence level and degrees of freedom in the statistic are related to the number of elements in the sample.

DISCUSSION

In determining the type of statistical analysis to be utilized it was recognized that variations observed in cell counts in each frame have several components:

1. Variations due to random nature of radioactive decay
2. Variations due to nonuniformity in the crystal detector, light pipe, photomultiplier system
3. Variations due to background and noise
4. Variations due to the patient.

When a uniform pool of radioactive material is utilized, patient variations are excluded. Attempts can then be made to measure the size of the remaining variations.

Two major branches of statistics provide mechanisms that may be used in analysis of the uniform pool data:

1. Classical sampling statistics. These tend to treat spatial and temporal aspects separately:
   a. Computation of means, variances, and confidence intervals
   b. Testing of hypothesis concerning population statistics using:
      (a) The Chi square test
      (b) Analysis of variance
      (c) Student's t

[redacted]

A SIMPLE BOLUS MOULD FOR INCREASING SURFACE DOSE WITH HIGH ENERGY BEAMS*

By R. V. SCHEELE, M.S., J. W. TURNER, Jr., FAYE HURTT, R.T.-T.,
and W. C. CONSTABLE, M.B., Ch.B., D.M.R.T.
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A SITUATION recently arose where the radiotherapist desired the skin-sparing effects of 8 MV. roentgen rays to be reduced to 1 cm. rather than the usual 2 cm. This was accomplished by devising a bolus mould composed of felt and wax. We believe that this idea, as described below, may be of benefit to other users of high energy beams.

MATERIAL AND METHOD

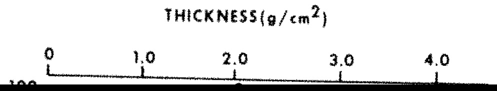
The patient to be treated was brought into the Division of Radiotherapy where a shell of the neck contour was made by applying a plaster of Paris bandage to the area of interest. The shell was allowed to harden and then removed from the patient. Its inner surface was coated with petroleum jelly and filled with a mixture of 3 parts stone plaster to 1 part water. This was allowed to harden forming a positive form of the patient contour. The plaster of Paris shell was then removed.

A piece of felt of appropriate thickness, depending on the degree of build-up desired, and precut to the approximate area desired, was allowed to soak in hot paraffin wax until it was saturated. This wax-saturated felt was applied to the outer surface of the positive form, shaped to its

[redacted]

0.91 ± 0.002 gm./cm.^{3}. This corresponds favorably to the density of paraffin wax ($\rho = 0.87$–0.91 gm./cm.^{3}) and also to the density of soft tissue ($\rho = 1.00$).[1,4]

Large sheets (10 × 10 inches) of the material were also made and the thickness determined. A 1N2482 Sarkes Tarzian diode detector, with an outside diameter of 0.4 cm., was placed in the center of a 10 × 10 cm. field at a constant source-to-probe distance of 100 cm. and the current measured with a Model 610C Keithley electrometer. The felt-wax plates were then placed directly above the diode probe and the dose rate determined. The dose rate, normalized to 1 for the maximum reading, was plotted against thickness of bolus material (Fig. 2). The curve shows that the maximum build-up occurs at 2.2 cm. of bolus material. Thus, to attain a peak absorbed dose rate at a depth of 1 cm. beneath the skin surface for 8 MV. roentgen



venient and economical to use. It conforms perfectly to the patient contour and can be

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MELTING POT FOR IRREGULAR SHAPED LEAD SHOT-THERMOPLASTIC SHIELDS

By YOSH MARUYAMA, M.D.,* DENNIS BURNS,† *and* LAWRENCE ESPY†
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INDIVIDUALIZED shielding using contoured lead shields is desirable under certain conditions; *e.g.*, in the radiotherapy of the mantle field in Hodgkin's disease. In 1969 a simple method for block construction useful for a clinic which does not have adequate shop facilities for casting or machining metallic lead was described.[2] Very simply, lead in the form of small shot can be placed in a thermoplastic matrix for the shielding of complex shapes such as the lungs.

MELTING APPARATUS

A simple apparatus designed as a melting pot dispenser of the matrix-lead shot mixture has now been constructed and used. The unit is essentially a stainless steel pot with walls lined with a heating coil. The heating element is controlled by a Virtis (model 51-TC) temperature controller with a variable temperature adjustment between 0–100°C. with a service mounted probe thermistor (50-4-D). The bottom of the pot is a conical funnel with a large outlet measuring 2 inches in diameter. A screw driven, rotatory plug controls the outlet. The unit may be set at about 80°C., which is the melting temperature of the thermoplastic.[2] Both the lead shot and plastic are placed in the melting pot and reduced to a molten slurry. Lead shot, being of much greater density, settles to the bottom of the pot and can be dispensed into the polyethylene or polystyrene mold forms which have previously been cut. In use, it has been found that further settling occurs in the mold so that the thermoplastic Rigidax rises to the top. The addition of a small amount of lead shot after initial pouring assures homogeneity of lead in the mold. After several hours, the shield is ready for use with or without removal from the polyethylene material.

In the past year, lung shield construction has become routine using this pot, and a patient seen one day can have his initial treatment on the afternoon of the next day with his individually constructed lung shield. The portal radiograph in the treatment position is the basis of shield construction; nonetheless, further fine adjustment is often desirable using variable block-surface-distance.[1]

A separate aspect of the melting pot relates to the re-utilization of the used blocks. After a period of time a clinic accumulates a number of previously used blocks which can simply be placed in the pot and melted down in several (3–4) hours to a recastable form. A final consideration relates to separation of the thermoplastic from the lead shot after the plastic compound has aged and lost its desirable features. This was accomplished by adding water, heating and repeated rinsing of the shot material. This has to be done only 1 or 2 times a year, especially if the temperature has been lowered to room temperature after each usage.

CONCLUSION

The pot described is of adequate size for this clinic, which treats about 50 to 75 mantle field patients per year. It would be adequate for 2 to 3 times this number. In a reduced size (≃50 per cent reduction in width and length scale) the unit would be useful for smaller clinics that treat a lesser number of patients annually, and do not have access to machining or casting facilities. The finished product may also be

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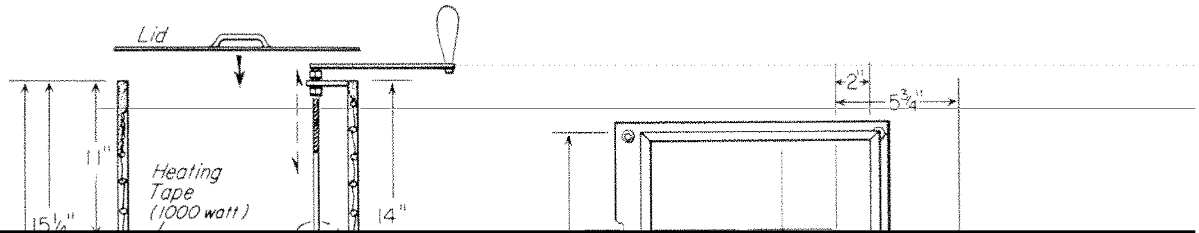

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UNTREATED AND RECURRENT CHROMOPHOBE ADENOMAS OF THE PITUITARY*

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THE treatment of chromophobe adenoma of the pituitary gland has been the subject of numerous reports.[2,4,6] These

[redacted]

who failed to return for observation, 2 with visual field defects refused treatment.

Recurrence as defined ~~for this study~~

Table I

PATIENTS WITH DIAGNOSIS OF CHROMOPHOBE ADENOMA (1931 to 1969)

Received radiation or surgical treatment, or both, at UCSF		
Single course of treatment		155
Multiple courses* of treatment		35
Deleted from present study	16†	
In present study of recurrence	19	
Only medical management, or no treatment at UCSF		29
Incidental finding at necropsy		40
Pituitary enlarged	7	
Pituitary normal size	33	
Total		259

* Does not reflect total number of recurrences as a few patients refused retreatment and some of the earlier patients were inadequately followed.

† Either primary radiation therapy or the retreatment was given elsewhere and could not be evaluated.

Table II

PATIENTS WITH PRESUMPTIVE DIAGNOSIS OF CHROMOPHOBE ADENOMA BUT INITIALLY UNTREATED

Eventual state unknown (no follow-up examination)	
Initially: Enlarged sella, but normal visual fields*	6
Initially: Enlarged sella and field defect	2
Follow-up observations available	
Initially: Enlarged sella with normal visual fields	
Sella became larger	3
3 treated at 5,† 6, and 15 years	
Visual field defects appeared	2
2 treated at 1,‡ and 5‡ years	
No change in sella or field	3
2 not treated; followed 4 and 20 years	
1 treated at 4 years (*symptoms* increasing)	
Initially: Visual field defects and enlarged sella	
Field defects became larger	5
4 treated at ½, 1½,‡ 3, and 5 years	
1 not treated; followed 8‡ years	
Sella and field defects increased	3
3 treated at 1,‡ 4,‡ and 5‡ years	

* One had hypopituitarism.

after 4 years because of persistent and increasing severe headaches. After completion of therapy 1 patient had cerebrospinal fluid rhinorrhea* and 2 others had moderate vi-

[redacted]

TABLE III

PRIMARY THERAPY: FRONTAL CRANIOTOMY WITH PARTIAL REMOVAL OF CHROMOPHOBE ADENOMA

Patient:	L	M	N	O	P	Q	R	S	T
Treatment	Surgery	Surgery	Surgery	Surgery	Surgery*	Surgery†	Surgery‡	Surgery*	Surgery*
Response	(same)	(better)	(better)	(better)	(better)	(better)	(better, normal)	(better)	(better)
Interval	2 yr.	7 yr.	2 yr.	1 yr.	3 yr.	3 yr.	8 yr.	4½ yr.	1 yr.
Status	(worse)	(worse)	(worse)	(worse)	(worse)	(worse)	(worse)	(blind)	(worse)
Treatment	3,800 r/ 16 da. 3,800 r/ 16 da.	Surgery	5,400 r/ 28 da. 2,800 r/ 180 da.	2,300 r/ 32 da.	Surgery 3,000 r/ 27 da.	Surgery 4,500 r/ 41 da.	4,000 r/ 45 da.	5,100 r/ 46 da.§	2,650 r/ 45 da. Surgery*
Response	(better)	(same)	(same)	(same)	(better)	(better)	(normal)	(?)	(normal)
Interval	10 yr.	12 yr.	4 yr.	8 yr.	12 yr.	6 yr.	7 yr.	5 da.	10 yr.
Status	(stable) DNRC	(stable)	(worse)	(stable)	(stable)	(stable) DNRC	(stable) DNRC	(dead)	(worse)
Treatment			Surgery*						Surgery 4,200 r/ 29 da.
Response			(?)						(better)
Interval			1 wk.						1 yr.
Status			(dead‖)						(stable)

* Cystic chromophobe adenoma.

† Also had meningioma of tuberculum sellae.

‡ Huge invasive adenoma.

§ General condition terminal; patient died 5 days after completion of irradiation; cause of death unknown.

‖ Surgical death.

(Comments in parentheses refer to visual field.) DNRC = Death not related to chromophobe adenoma. Radiation doses are cited as r to pituitary in given number of days; *e.g.*, 3,800 r/16 da.

TREATMENT OF RECURRENT CHROMOPHOBE ADENOMA

Recurrent chromophobe adenomas in 19 patients received therapy after primary, presumably definitive therapy had been given for the same disease. One had received radiation therapy only. Craniotomy with partial removal of the adenoma had been performed in 9. Since these patients did not receive postoperative radiation therapy, their initial therapy might be considered as incomplete. The other 9 patients had undergone surgical decompression plus postoperative radiation therapy. In all patients the radiation therapy was given either at the University of California Medical Center or at a nearby institution. Detailed data relative to the treatment were available on all patients included in the study. Conclusions based on a single case are unsatisfactory. Hence, the one tumor initially irradiated only, which at craniotomy 2 years later and at postmortem examination 6 months postoperatively proved to be a chromophobe adenocarcinoma, will not be considered further.

In this material, the interval between primary and secondary treatment averaged 3.5 years (range from 1 to 8 years) when the earlier therapy was surgery alone (Tables III and IV). The average interval after combined treatment, surgery plus postoperative irradiation, was 8.9 years (range from 2½ to 17 years) (Table V). It appears that in those tumors destined to recur, the irradiation may have delayed the recurrence.

At the time of the first treatment, of those tumors initially treated by surgery alone, a locally invasive adenoma was described in 1 patient (B). By contrast, 5 of the 9 with primary combined treatment had

TABLE IV

SUMMARY OF TABLE III

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TABLE V

PRIMARY THERAPY: FRONTAL CRANIOTOMY WITH PARTIAL REMOVAL OF ADENOMA AND RADIATION THERAPY

Patient:	C	D	E	F	G	H	I	J	K
Treatment	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery	Surgery

[redacted]

TABLE VI

SUMMARY OF TABLE V

in 4 and stabilized fields in 3. In 8 others, the combined retreatment by surgery and radiation therapy resulted in improvement

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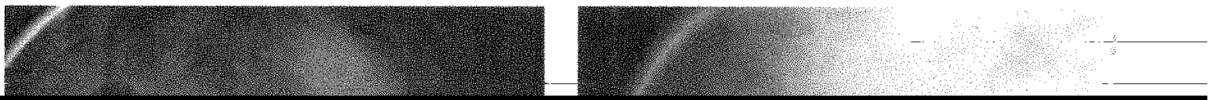

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TABLE I

CLINICAL MATERIAL

	Hysterectomy	No hysterectomy
Choriocarcinoma	5	—
Chorioadenoma destruens	7	—
Hydatidiform mole	3	16
Total	15	16

with a No. 20 scalp-vein needle and flushed with heparinized saline to avoid clotting. Then, micropaque (barium sulfate suspension) was injected slowly. Roentgenograms were taken at each injection of 1.5 to 2.0 cc. until considerable resistance to the injection was encountered. Usually a total of 7 to 8 cc. of contrast medium was required. Following fixation of the uterus in 10 per cent neutral buffered formalin solution for 7 to 10 days, cross sections of uteri 0.5 to 1.0 cm. thick were made and fourfold magnification angiograms were taken in the same manner as described above.

The major angiographic findings were classified as follows: (1) increased myometrial vascularity; (2) presence of tumor vessels; (3) central and peripheral vascular pooling; (4) central avascularity; and (5) early venous filling. These angiographic abnormalities were compared with the findings of magnification angiography of the removed uteri. Analysis was also made to answer the question whether or not pelvic angiograms were suggestive of any particular histologic type of trophoblastic tumors.

RESULTS

HYDATIDIFORM MOLE

Nineteen patients with hydatidiform mole were examined by pelvic angiography. Seventeen of these were sent to our clinic following a mole abortion. The remaining 2 patients visited our hospital before removal of the uterine contents, and eventually underwent a curettage at our hospital. Three of the 19 patients underwent a hysterectomy, since the urinary human chorionic gonadotropin titer did not return to the expected low level within 3 weeks following mole abortion. Magnification angiography was performed on these 3 resected uteri.

Pelvic angiograms usually showed slight to moderate dilatation of the uterine arteries with the diameter measuring 1 to 2 mm. in the proximal portion. One or both ovarian arteries were enlarged in 10 of 19 patients. The myometrial arteries were slightly dilated and seen as spiral vessels in all patients (Fig. 1, *A* and *B*). Space-occupying lesions with vascular pooling or central avascularity were not demonstrated.

Two patients, whose uteri were enlarged without signs of a fetus, showed irregular vascular spaces in addition to enlarged, stretched uterine, ovarian and myometrial arteries (Fig. 2, *A* and *B*). The irregular vascular spaces were thought to be present in the intervillous spaces of the tumor. There were multiple round filling defects with sharp margins surrounded by vascular spaces. These were thought to correspond to the cystic degeneration of the villi. Evacuation of the uteri confirmed the angiographic diagnosis.

Magnification angiography of the 3 resected uteri showed multiple, fine, spiral myometrial arteries (Fig. 1, *C* and *D*). No vascular pooling, avascular lesions or early venous filling were demonstrated, corresponding to the findings of pelvic angiography.

INVASIVE TROPHOBLASTIC TUMORS

There were 12 patients who were classified under this heading. Hysterectomy was performed on all patients. Seven patients were diagnosed as having chorioadenoma destruens on histologic examination of the resected uterus, while a diagnosis of choriocarcinoma was made in 5 patients. Two resected uteri from patients with choriocarcinoma were not available for magnification angiography. Thus, evalua-

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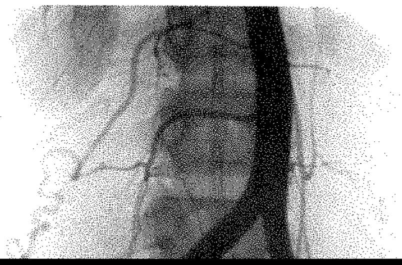

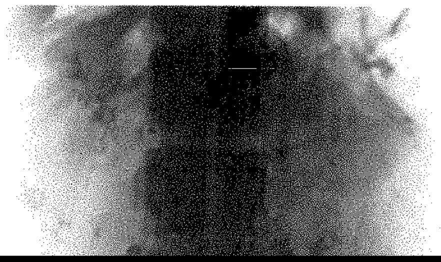

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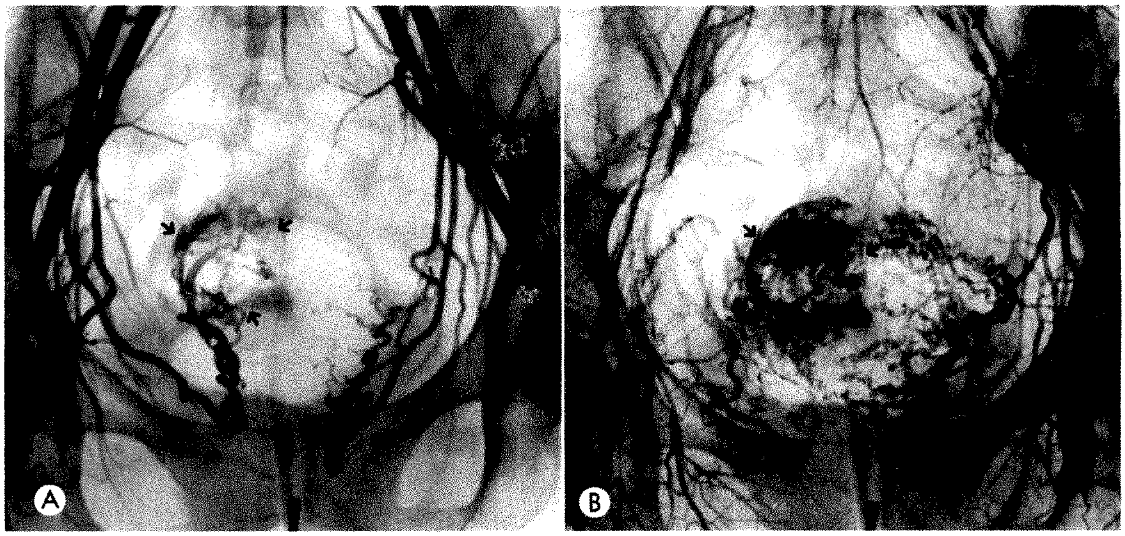

FIG. 4. Chorioadenoma destruens in a 47 year old Japanese female.
(*A*) Arterial phase of pelvic angiogram. Both uterine arteries and myometrial vessels are enlarged. There is a space-occupying lesion in the right fundus which is surrounded by tortuous myometrial vessels. They simulate tumor vessels (arrows). (*B*) Capillary phase of the angiogram demonstrates central avascularity of the lesion with peripheral vascular pooling (arrows). Spiral myometrial vessels are well shown.

cally demonstrated in 7 patients, and in 6 of them these changes were confirmed by magnification angiography (Fig. 5, *A–C*). An incorrect diagnosis was made in 2 patients by pelvic angiography. In the other 2 patients central avascularity was not present on either the pelvic or magnification angiograms.

(5) *Early venous filling.* The veins around the lesion draining into the ovarian or iliac veins were usually demonstrated in the late capillary phase of pelvic angiography. Slightly earlier filling of the veins was seen in 2 patients on the pelvic angiograms, and in 1 of them this was confirmed by the magnification angiogram.

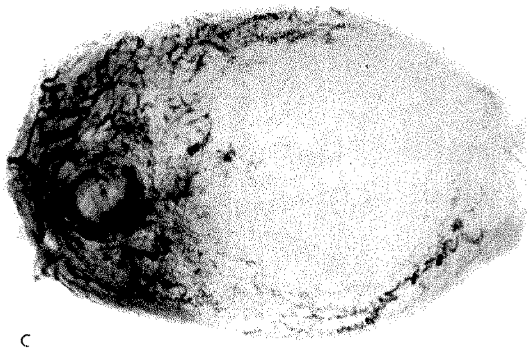

FIG. 4. (*C*) Magnification angiogram of the uterus shows lobulated central avascularity and peripheral vascular pooling. No definite tumor vessels are noted.

There was no case which showed arteriovenous fistula in or around the lesions. The filling of the veins was usually via the capillaries rather than arteriovenous fistulae on both the pelvic and magnification angiograms (Fig. 5, *A–C*).

DIFFERENTIATION OF CHORIOADENOMA DESTRUENS AND CHORIOCARCINOMA BY ANGIOGRAPHIC FINDINGS

It was difficult to differentiate these 2 entities by pelvic angiography (Table III). Central vascular pooling was present in 4 of 7 cases with chorioadenoma destruens, and this finding was not present in any of 3 choriocarcinomas. Peripheral vascular pooling was present similarly in both entities. There was central avascularity in 2 of 3 cases of choriocarcinoma, while this finding was present in 5 of 7 cases of chorioadenoma destruens. Enlarged myometrial vessels, tumor vessels and early

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ings of injection studies of the operative specimen. It has been mentioned that good results with such studies depend upon the quality of the contrast material, duration and pressure of the injections, the state of the specimens, etc.[11,15] Serial filming with fractionated injections of 2 to 3 cc. of contrast medium such as used in this study seems to produce angiograms of nearly a natural state of the vasculature. In fact there has been fairly good correlation between the pelvic angiography and the magnification angiography in this study, enabling us to evaluate the findings of pelvic angiography by this technique.

There was a high conformity of the pelvic angiographic findings with those of magnification angiography of the removed uteri in demonstration of increased myometrial vessels, peripheral vascular pooling, central avascularity, and central vascular pooling. However, the tumor vessels which were frequently suggested on pelvic angiography were not present within the trophoblastic lesions of most cases on magnification angiograms.

In spite of some difficulties encountered in interpretation, it was not hard to differentiate the benign process from the malignant trophoblastic tumors, since patients with hydatidiform mole following curettage showed merely increased myometrial vessels without other angiographic abnormalities. There was no evidence of space-occupying lesions within the uterus. In contrast, malignant trophoblastic tumors showed findings such as vascular pooling, central avascularity and enlarged draining veins.

It has been stated that choriocarcinoma can be differentiated from chorioadenoma destruens on pelvic angiograms,[5,10] since the former frequently shows central avascularity with peripheral vascular pooling. Brewis and Bagshawe[5] reported that 7 of 8 cases with choriocarcinoma revealed only slight enlargement of the uterine artery and slight stain within the lesion with no vascular pooling or vein visualization. In our series, the combination of minimal peripheral vascular pooling and central avascularity was suggestive of choriocarcinoma, while central vascular pooling was more consistent with chorioadenoma destruens. Choriocarcinoma frequently showed minor degrees of vascularity within the uterus as well as in the lesion.

By pelvic angiography it is possible to detect development of malignant trophoblastic tumors following delivery or curettage of benign hydatidiform mole. Hendrickse *et al.*[8] reported that the presence of malignant trophoblastic deposit could be detected by angiography even before the excretion of chorionic gonadotropin reached the level found in normal pregnancy. On the contrary, there has been a case in the present study which developed malignant trophoblastic disease in the absence of angiographic abnormalities. Such a case might be unusual.

There have been several reports which emphasize the significance of pelvic angiography in evaluating a favorable response of trophoblastic tumors to chemotherapy.[6,9,14] This has been confirmed in our experience, but there were some cases in which abnormal circulation remained at the completion of the chemotherapy with clinical curative effect.[8] This may be due to the fact that resolution of the vascular abnormalities requires more time. Persistent arteriovenous fistulae after clinical cure of the disease have been described.[7,12,16] Furthermore, abnormal pelvic angiograms may revert to normal in spite of persistent disease in the uterus.[5] Thus, response of the disease to chemotherapy and the findings of the pelvic angiograms do not always coincide.

Pelvic angiography is frequently valuable for differentiation of trophoblastic tumors from missed abortion or normal pregnancy. In missed abortion, there are irregular vascular spaces arranged around a central avascularity,[2,5] while the angiographic appearances of normal pregnancy are diffuse, prolonged opacifications in a localized area of the uterus, which represent filling of the placental sinuses.[2] Pelvic angiography can also be used for the differential diagnosis of other gynecologic tumors.[13]

In order to avoid superimposition of vessels on the uterus, we have applied traction on the uterine cervix. This tech-

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3. Borell, U., Fernström, I., Moberger, G., and Ohlson, L. The Diagnosis of Hydatidiform Mole, Malignant Hydatidiform Mole and Choriocarcinoma, with Special Reference to the Diagnostic Value of Pelvic Arteriography.

IMPROVED PHOTOGRAPHIC LEAK TEST SYSTEM FOR SEALED SOURCES OF RADIUM 226[*]

By P. H. BEDROSIAN, Ph.D.,[†] *and* E. L. SENSINTAFFAR
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TWO photographic methods for leak testing sealed sources of radium 226 have been documented.[1,2] Both procedures use high-speed photographic emulsion to detect light produced by interactions between a ZnS screen and alpha particles emitted by leaking sources. The Polaroid Land 4×5 film system, which includes individually packeted photographic film and film holder, is used in these methods. The film packet includes its own developing solution, and exposure of the film is made by sliding off the protective packet envelope. Exposure and development of the film is achieved by use of the film holder. For these methods, orientation of the source, ZnS screen, and photographic emulsion required that the source be placed within the source chamber of the device prior to exposing the film. Since the film is usually uncovered manually while the source is only a few centimeters away in the chamber, unnecessary radiation exposure to the upper part of the body, particularly to the hands and arms of the operator, may occur. While careful handling and good practice may reduce personnel exposure, minimal exposure would occur if the test procedure were reversed, *i.e.*, the film uncovered then the source placed into the chamber. To achieve this, the photographic unit for detecting radon was modified.[2] The essential aspect of the modification lay in fabricating a device in which the source chamber could remain open to light without fogging the film if it were in the exposed position.[*] Figure 1, *A* and *B* depicts schematically the main

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[*] As in the other referenced photographic methods, the modified version uses Polaroid Land film Type 57 ASA 3000. Use of the Polaroid equipment does not constitute endorsement by the U.S. Public Health Service.

[*] From the Department of Health, Education, and Welfare, Public Health Service, Bureau of Radiological Health, Southeastern Radiological Health Laboratory, Montgomery, Alabama.

[†] Present Address: Environmental Protection Agency, Office of Research and Monitoring, Radiation Research, Research Division, Rockville, Maryland.

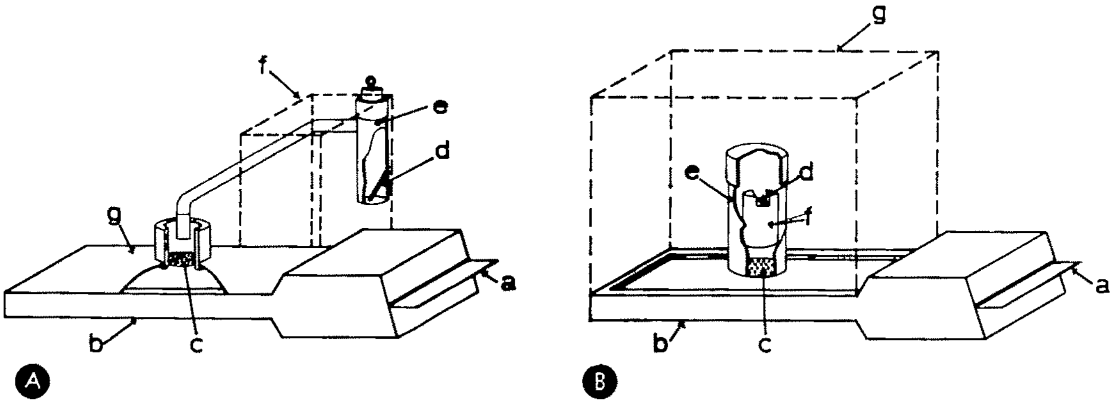
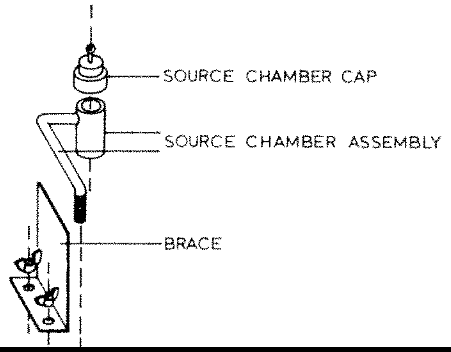


Fig. 1. (*A*) Diagram depicts modified device which can be loaded with radioactive source while film is in exposed position. (*B*) Diagram shows unmodified unit which permits film to be exposed only after source is in the capped chamber. a = film and packet; b = film holder; c = phosphor; d = source; e = source chamber; f = lead shield; and g = light tight cover.

components of the device with and without the modification.

The modified unit consists of a bent tube which leads radon from the chamber to the ZnS phosphor. Bends in the tube permit radon to reach the phosphor, but allow essentially no light to reach the film while the source chamber is open. In this manner, the operator leaves the chamber open, uncovers the film and contacts the



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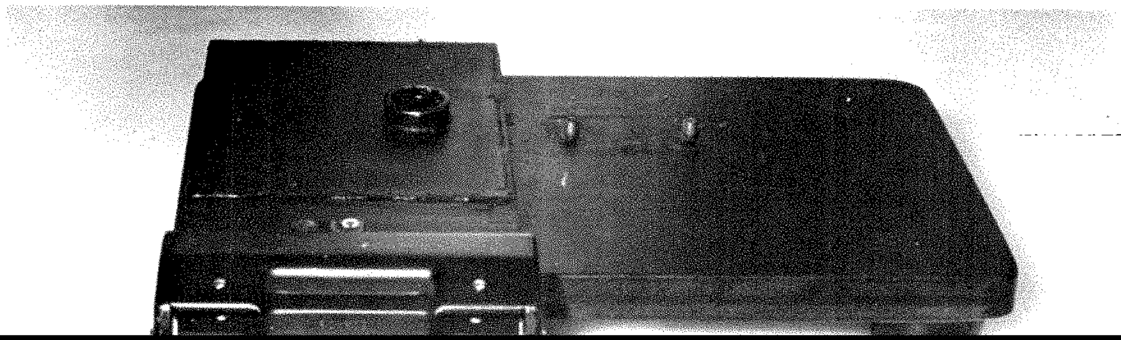

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Fig. 3. Major components of the disassembled unit show (*A*) the mounted film holder; (*B*) the 4 inch thick lead shield; and (*C*) the ZnS window-source chamber assembly.

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for field applications. It may be connected to storage safes and calibrated for testing sources while they remain in storage.

Leak testing of large numbers of sources by this technique may be safely done on a daily, weekly, or longer basis.

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SYNCHRONOUS SHIELDING AND FIELD SHAPING FOR MEGAVOLT IRRADIATION OF ADVANCED CERVICAL CARCINOMA*

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THE importance of external pelvic irradiation in patients with moderately advanced carcinoma of the cervix is readily apparent. Intracavitary radium or its substitutes cannot safely deliver a cancericidal dose to all of the involved tumor volume. This potential tumor volume includes not only the usually involved utero-cervico-vaginal and medial parametrial complex of tissues, but also portions of the broad ligament and the pelvic lymph nodes. Optimal irradiation of this irregular tumor volume must be accomplished without over-irradiation of uninvolved adjacent structures such as skin, bladder, rectum, small intestine, and proximal portions of the femora.

Over the past decade this laboratory has been involved particularly in devising megavolt radiation techniques utilizing patient rotation, synchronous external field shaping and special protection.[1–3,5–7] Takahashi[4] has described one method of synchronous field shaping applied to carcinoma of the cervix.

TECHNIQUE

A 2 million volt Van de Graaff generator is directed horizontally toward a patient standing on a rotating platform with rotating metal absorbers placed in the path of the x-ray beam. Absorbers for field shaping conform the shape of the x-ray beam to the known and estimated tumor volume, thereby protecting adjacent normal regions by exclusion: absorbers for synchronous shielding further reduce the dose to critically sensitive normal tissues.

Figure 1 illustrates this technique on cross section. Oval blocks, one on each side of the beam, rotate at the same number of revolutions per minute, *i.e.*, synchronously, as the patient. The upper diagram shows the largest separation between these absorbers for field shaping. The middle and lower diagrams represent consecutive rotations of 45°. The lower diagram shows the smallest separation between the absorbers and consequently the smallest beam width. Rotating through 360°, the field shaping absorbers contour the x-ray beam to form an elliptical dose distribution within the patient.

As shown in Figure 1, the special absorbers for rectum and bladder are placed in the path between the x-ray source and these structures. These absorbers are also synchronized with the rotation of the patient; thus, rectum and bladder are continually shadowed with a resultant reduction in radiation dose. This synchronous shielding changes the elliptical dose con tour produced by the field shaping to a dumbbell shape.

Figure 2 shows an oblique view of patient and apparatus. Paired dotted lines at top

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FIG. 2. Oblique view of patient and equipment for rotational therapy with synchronous shielding and field shaping. X-ray beam is directed horizontally toward patient and modified by field shaping absorbers to produce continuously irradiated volume (CIV). Absorbers for synchronous shielding protect portions of small intestine (I), bladder (B), and rectum (R).

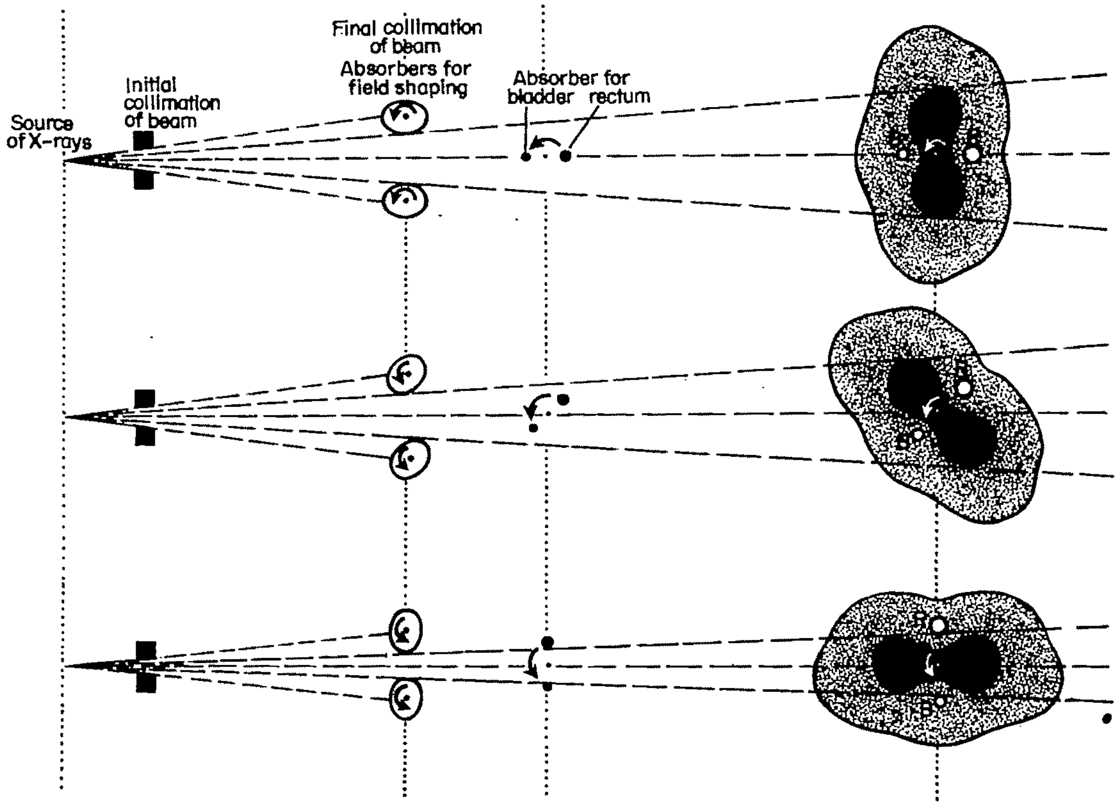
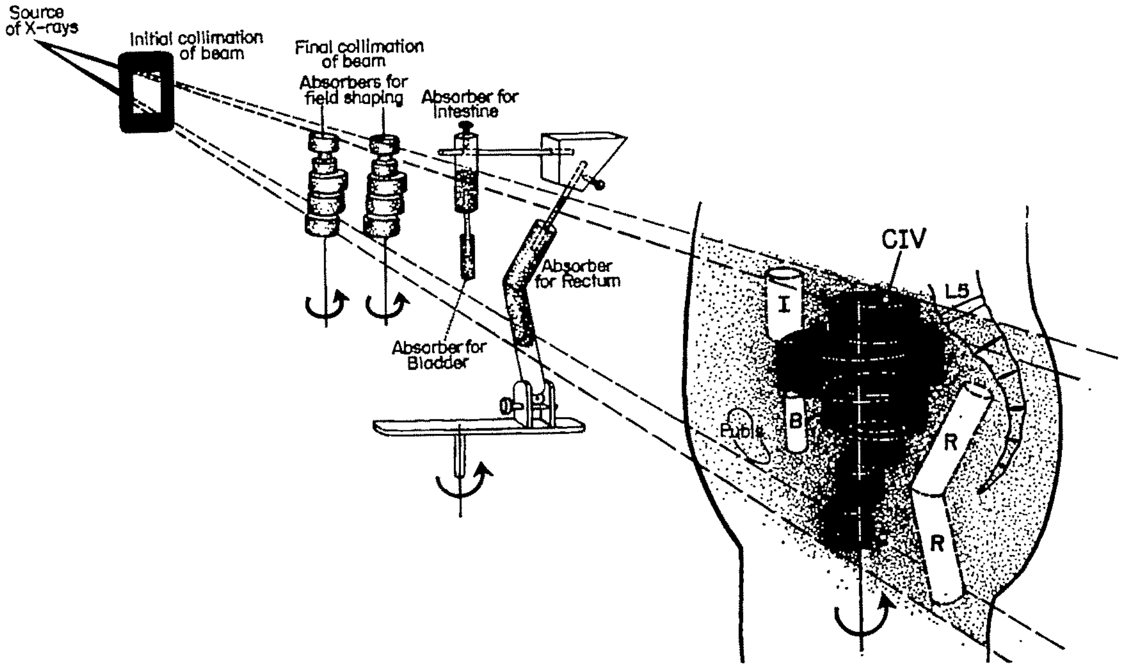

Final collimation of beam
Absorbers for field shaping
Absorber for bladder rectum
Initial collimation of beam
Source of X-rays
R
B
R
B
R
B

Fig. 1. Three representations of a cross section showing technique of rotational therapy with synchronous shielding and field shaping. Absorbers for field shaping and shielding rotate at same rate as patient. Field shaping restricts higher radiation levels to limited volume. Synchronous shielding further reduces dose delivered to protected portions of bladder (B) and rectum (R), and changes elliptical dose contour produced by field shaping to dumbbell shape.



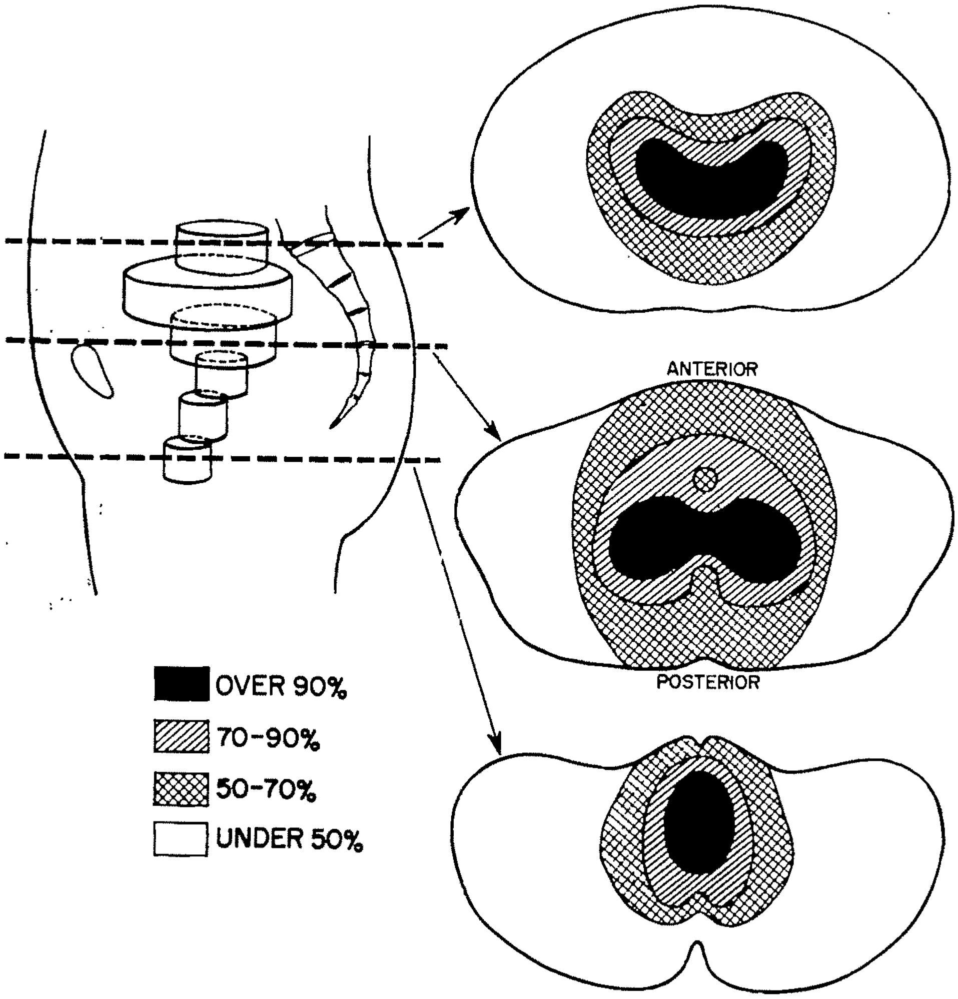


Fig. 3. Dose distribution for 3 pelvic cross sections resulting from rotational therapy with synchronous shielding and field shaping. Potential tumor volume is contained inside zone receiving more than 90 per cent of tumor dose. Contour of this maximally irradiated zone varies in size and shape at different levels corresponding with clinical estimated tumor volume.

and bottom delineate the height of the field of treatment extending from the bottom of the fifth lumbar vertebra to the inferior margins of the ischia. The variability in shape and location of the tumor volume at different levels requires 6 pairs of appropriately shaped brass blocks to define the contour. While the field shaping partially protects the bladder, rectum, and small intestine, additional protection is obtained with rotating absorbers for synchronous shielding. These absorbers may be lead, brass, or other material depending on the absorption characteristics desired, and of varying size depending on the relative location of bladder, rectum, and small intestine. The relative spatial configuration of these 3 absorbers to each other is adjustable.

The shape of the rectal absorber approximates the anteroposterior curve of the anorectal canal below the second sacral segment, providing protection to the anterior wall throughout 360° rotation and some protection to the posterior wall. The lateral deviations of the rectum are of minor consequence. The additional protection from the synchronous field shaping

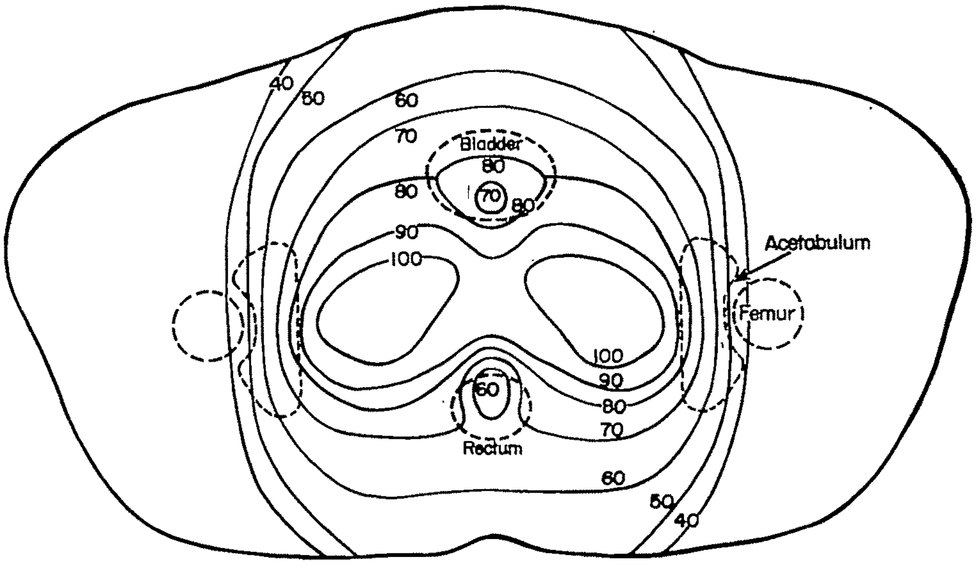


Fig. 4. Isodose curves for pelvic cross section at the level of the superior margin of symphysis pubis. Numbers shown represent percentages, with potential tumor volume contained in 90 per cent isodose curve. Bladder contour represents lower third of bladder with major part lying superior and more anterior and receiving from 60 to 85 per cent of tumor dose.

lowers the dose to the posterior wall of the rectum. Most of the protection for the bladder and small intestine is provided by the field shaping absorbers. The bladder and small intestinal absorbers contribute additional protection, especially to the posterior wall of the bladder. Instead of shielding the walls of the bladder and rectum immediately adjacent to the tumor, the absorbers could be adjusted to protect the anterior mucosa of the bladder and the posterior mucosa of the rectum, thereby simplifying the alignment.

DOSIMETRY

Measurements were made from films sandwiched at several levels within a masonite phantom. The film had been calibrated with a photodensitometer and thermoluminescent dosimeters, giving the correlation between film density and exposure dose.[1]

Figure 3 shows dose distributions for 3 pelvic cross sections. A tumor dose homogeneity of ±10 per cent was obtained for the entire tumor volume with the tumor dose designated as 100 per cent. The contour of the maximally irradiated zone (receiving more than 90 per cent of the tumor dose) varies in size and shape at different levels corresponding with the clinically estimated tumor volume.

Figure 4 shows the cross-sectional dose distribution at the level of the superior margin of the symphysis pubis. The potential tumor volume was irradiated laterally out to the pelvic wall, with the head of the femur receiving 30 to 50 per cent, the bladder 75 to 85 per cent, and the rectum 60 to 70 per cent of the tumor dose. Measurements at other pelvic levels gave the bladder dose range as 60 to 85 per cent and the small intestinal dose range as 30 to 70 per cent of the tumor dose.

SUMMARY

A method of synchronous shielding and field shaping for megavolt x-ray rotational therapy of carcinoma of the cervix is presented.

Metal absorbers protect the bladder, rectum, and small intestine during 360° rotation while other blocks contour the shape of the x-ray beam to the known and estimated tumor volume.

Tests with a masonite phantom indicate

a tumor dose homogeneity of ± 10 per cent. The head of the femur received 30 to 50 per cent, the bladder 60 to 85 per cent, the rectum 60 to 70 per cent, and the small intestine 30 to 70 per cent of the tumor dose.

This technique optimizes the tumor dose relative to the adjacent normal tissue dose, thereby permitting higher radiation levels to be delivered to the entire tumor by external irradiation alone.

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not vary appreciably with field area, but it will be necessary to retain the phantom scatter ratio $S(A_m, E)/S(A_d, E)$ for energies lower than 1 MV.

The results in Table III show a similar independence of T_{mr} over source-to-axis distance as was shown by Johns *et al.*[5] for T_{ar}. This reduces the relationship for T_{mr} at higher energies to

$$T_{mr}(d, A_d, E) = \frac{D_d}{D_{dm}} = \frac{P}{100}\left(\frac{F+d}{F+d_m}\right)^2.$$

This relation for T_{mr} does not include the backscatter factor necessary for evaluating T_{ar} which is given by Johns and Cunningham[6] as

$$T_{ar}(d, r_d, Q) = \left(\frac{F+d}{F+d_m}\right)^2 B(r_m, Q)\,\frac{P}{100}\,(d, r_m, F, Q).$$

Holt *et al.*,[3] however, think that there is a need to retain the factor $S(A_m, E)/S(A_d, E)$ (the ratio of two scattered components) at 6 MV even if it is small. With the simpler relation for T_{mr}, we feel that there is no need to evaluate scatter functions, and percentage depth dose tables are sufficient for calculating T_{mr}.

It should be remembered, however, that D_{dm} is the dose at point P with only sufficient depth of tissue overlying P as is necessary for maximum build-up. Hence, the machine should be calibrated taking this into consideration.

T_{mr} can also be used to determine the percentage depth dose at different SSDs (source skin distance) in the following way.

Equation 1 can be re-written as

$$P(d, A_m, F_1, E) = 100T_{mr}(A_1, d, E)\cdot\left(\frac{F_1+d_m}{F_1+d}\right)^2\cdot\frac{S(A_1, E)}{S(A_m, E)},$$

where

$$A_1 = A_m\left(\frac{F_1+d}{F_1}\right)^2;$$

similarly

Table III

EXPERIMENTAL DETERMINATION OF T_{mr} AT VARIOUS SOURCE-TO-AXIS DISTANCE (SAD)

SAD (cm.)→ Depth in cm. ↓	80 T_{mr}	100 T_{mr}	120 T_{mr}
2	1.000	1.000	1.000
4	0.965	0.970	0.970
6	0.918	0.919	0.919
8	0.869	0.866	0.865

$$P(d, A_m, F_2, E) = 100T_{mr}(A_2, d, E)\cdot\left(\frac{F_2+d_m}{F_2+d}\right)^2\cdot\frac{S(A_2, E)}{S(A_m, E)},$$

where

$$A_2 = A_m\left(\frac{F_2+d}{F_2}\right)^2;$$

dividing we get

$$\frac{P(d, A_m, F_2, E)}{P(d, A_m, F_1, E)} = \frac{T_{mr}(A_2, d, E)}{T_{mr}(A_1, d, E)}\cdot F_f\cdot\frac{S(A_2, E)}{S(A_1, E)},$$

where

$$F_f = \left(\frac{F_2+d_m}{F_2+d}\right)^2 \Big/ \left(\frac{F_1+d_m}{F_1+d}\right)^2$$

(Mayneord and Lamerton).[8]

For SSDs between 80 and 120 cm., $S(A_2, E)/S(A_1, E)$ will be a negligible factor for high energies. This reduces the equation to

$$\frac{P(d, A_m, F_2, E)}{P(d, A_m, F_1, E)} = \frac{T_{mr}(A_2, d, E)}{T_{mr}(A_1, d, E)}\cdot F_f.$$

The factor F_f corrects the depth dose for inverse square law effect, while the ratio of the two tissue maximum-dose ratios correct for the difference in the solid angle of the beam.

The experimentally determined T_{mr}'s are in close agreement with those calculated from percentage depth dose tables.

SUMMARY

The idea of tissue maximum-dose ratio is considered better than tissue air ratio and

tissue phantom ratio for calculating dose at high energies.

The T_{pr}'s for 8 MV x rays have been calculated from percentage depth dose tables and are tabulated.

The calculated data have been compared with experimental measurements, and are found to be in good agreement.

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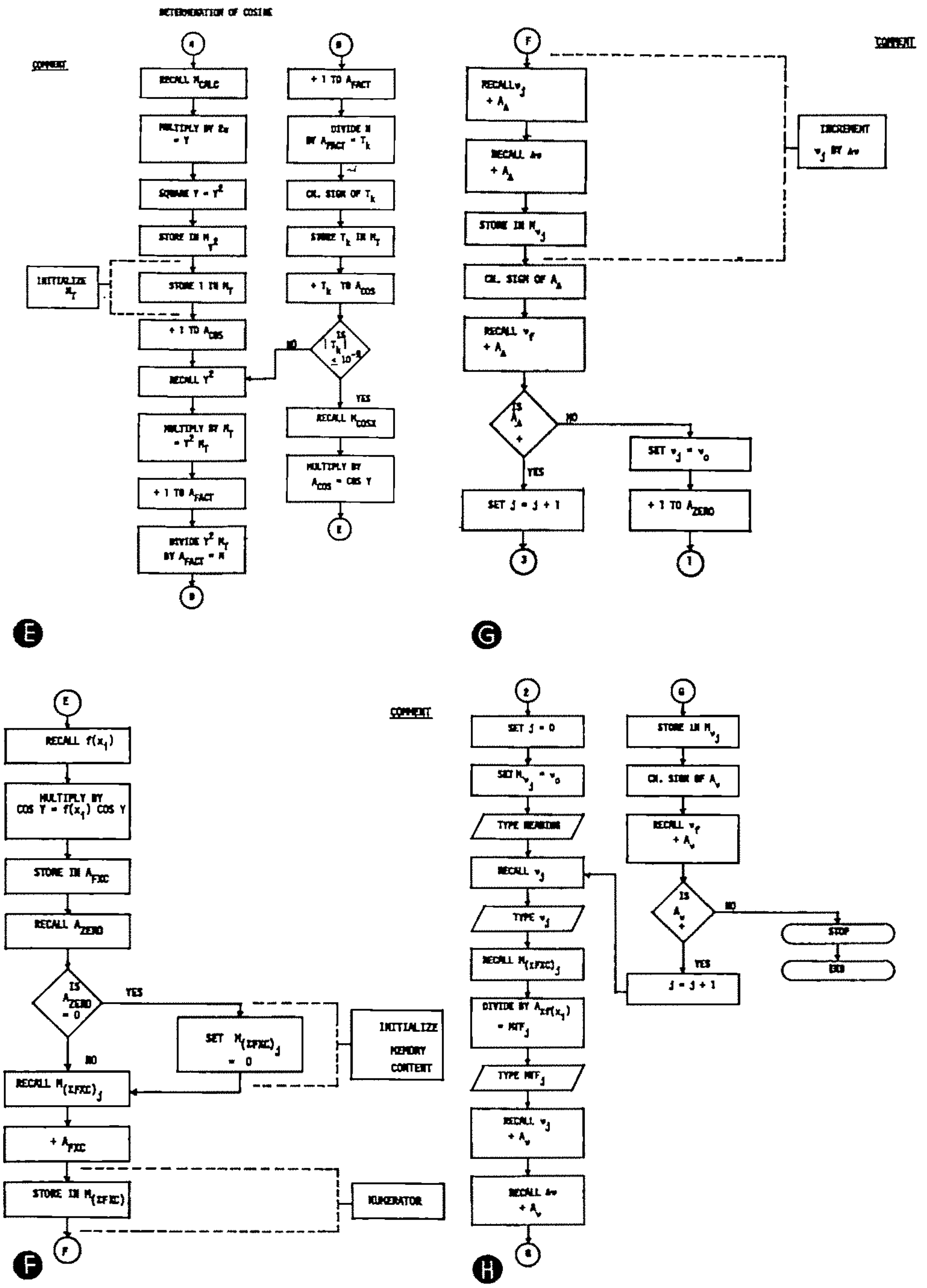

chart of the algorithm.

source. This determination is limited only by the number and accuracy of the data points obtained in counting the line source. It is evident from consideration of the cyclic nature of the cosine expression that the maximum spatial frequency for which the MTF can be determined is a function of the interval between data points (Equation 5).

The ease of determining the performance

TABLE I

DEFINITION OF SYMBOLS EMPLOYED IN THE ALGORITHM

Symbol	Meaning
A. *Basic Symbols*	
Z	Distance from detector to plane of LSF
ν_0	Initial spatial frequency
$\Delta\nu$	Incremental spatial frequency
ν_f	Final spatial frequency
x_i	Abscissal distance on LSF plane
$F(x_i)$	Value of LSF at x_i
Y	$2\pi\nu x$
T_k	Term in cosine expansion series
B. *Memories*	
Memory	Content
M_{ν_0}	ν_0
$M_{\Delta\nu}$	$\Delta\nu$
$M_{\nu f}$	ν_f
$M_{\nu j}$	Current value of ν
M_{xi}	x_i
$M_{F(x_i)}$	$F(x_i)$
$M_{\nu x}$	Current value of νx
$M_{\cos s}$	A multiplier which rectifies the sign of $\cos Y$
M_{calc}	The first quadrant equivalent value of νx to be used in cosine calculation
M_{Y^2}	Value of Y^2 for computation of cosine
M_T	T_k
$M_{(\Sigma FXC)_j}$	Numerator for each value of ν
C. *Adders*	
Adder	Use
A_{zero}	Indicator to initialize memory to zero
$A_{\nu x}$	Reduce the value of νx to equivalent first quadrant angle
$A_{\cos}$	Accumulate terms of cosine series
A_{Fact}	Determine denominator of cosine term T_k
A_{FXC}	Sum the individual values of $F(x_i)$ $\cos 2\pi\nu x_i$ for constant ν
A_{Δ}	Increment ν by $\Delta\nu$
$A_{\Sigma F(x_i)}$	Determine denominator of Equation 2
A_ν	To determine when calculations for ν_f have been completed

characteristics of a typical imaging system is illustrated by the example in this paper. The line source was constructed and 3 line spread functions determined in 1 morning;

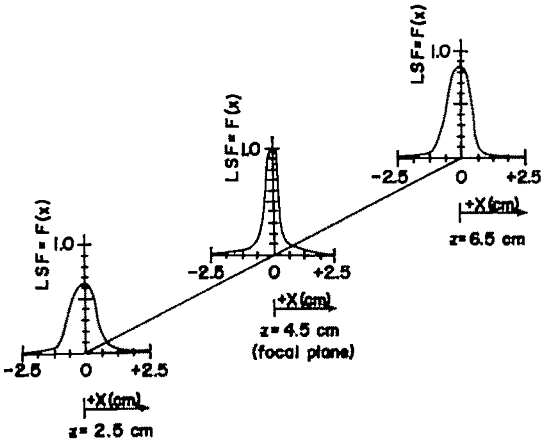

FIG. 2. Normalized Line Spread Function (LSF) at 3 source to collimator distances.

TABLE II

SAMPLE CALCULATOR OUTPUT

Modulation Transfer Function (MTF) *vs.* Frequency for Line Spread Function (LSF) $Z=4.5$

x	$F(x)$
.00	13638.
.32	1087.
.63	797.
.95	415.
1.27	227.
1.59	153.
1.90	84.
2.22	52.
2.54	35.
.32	10240.
.63	1764.
.95	650.
1.27	183.
1.59	154.
1.90	139.
2.22	101.
2.54	95.
Frequency	*MTF*
.00	1.0000
.20	.8645
.40	.6878
.60	.5176
.80	.3822
1.00	.2777
1.20	.2083
1.40	.1525
1.60	.1379

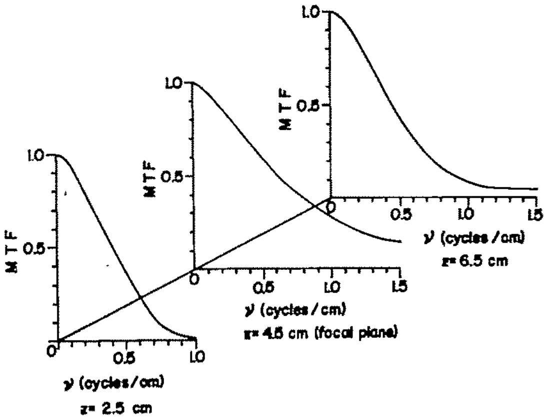

Fig. 3. Modulation Transfer Function (MTF) *vs.* spatial frequency at 3 source to collimator distances.

the corresponding modulation transfer functions were computed that afternoon. Hence, this program provides a very convenient and inexpensive method for the determination of performance characteristics of imaging devices by almost every nuclear medicine laboratory.

SUMMARY

An algorithm has been developed for the automated determination, by an iterative technique, of the modulation transfer function for the line spread function data.

The logic presented herein has been employed to develop a technique for this determination using a programmable desk-top calculator.

Examples illustrating the utility of the program are also presented.

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DO HIGH DETAIL SCREENS ALWAYS YIELD BETTER RESOLUTION THAN HIGH SPEED SCREENS?*

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IN MEDICAL radiography, it is commonly assumed that high detail screens yield better resolution than high speed screens. For this reason, the former are sometimes preferred to the latter, in spite of their need for increased radiation exposure.

However, can it be said that high detail screens always yield better resolution than high speed screens? When stationary objects are imaged, the answer to this question is certainly in the affirmative if the x-ray tube has an infinitely small focal spot, as shown on the left side of Figure 1, or if the objects being imaged are in intimate contact with the film-screen combination, as shown on the right. In either of these 2 special cases, the dimensions of the focal spot do not have any effect on resolution, thus the modulation transfer function (MTF) of the film-screen combination is the only parameter governing resolution. The MTF of high detail screens is, in general, superior to that of high speed

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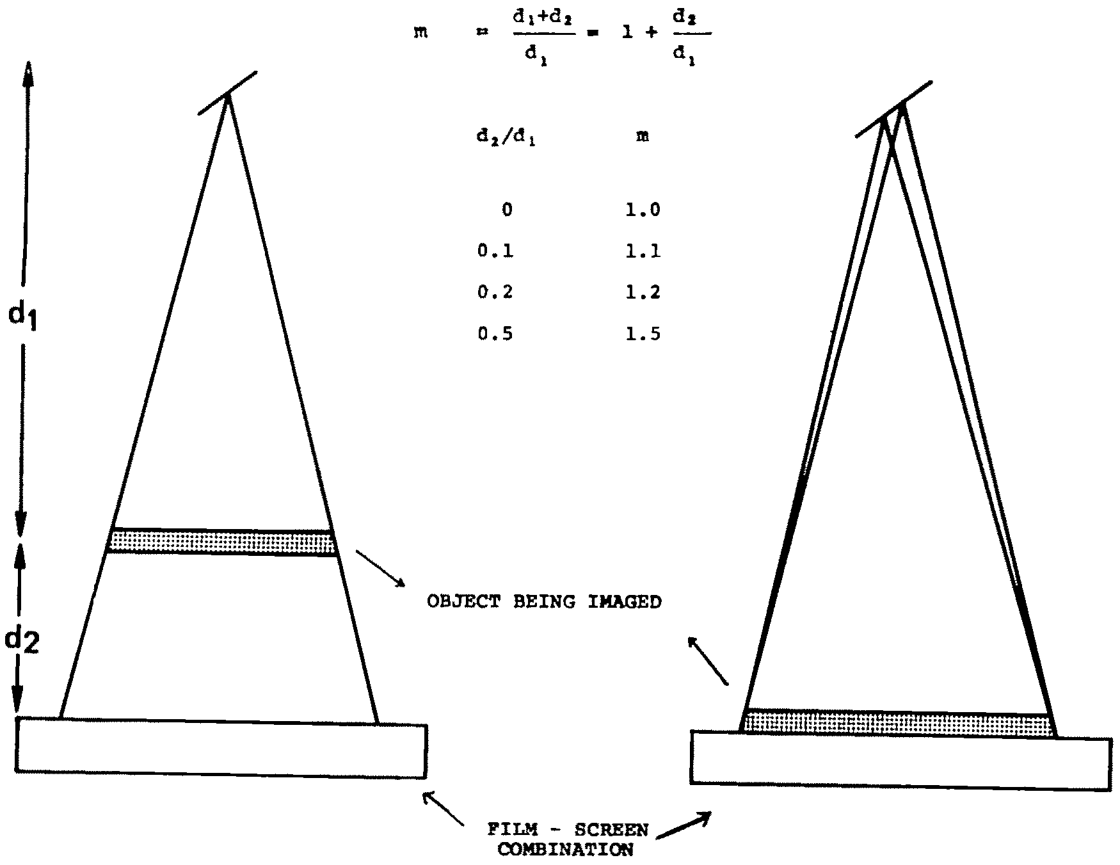

$$m = \frac{d_1+d_2}{d_1} = 1 + \frac{d_2}{d_1}$$

d_2/d_1	m
0	1.0
0.1	1.1
0.2	1.2
0.5	1.5

Fig. 1. Diagram illustrating the 2 special cases when high detail screens undoubtedly yield better resolution than high speed screens (see text for detailed explanation).

screens and hence in cases where the focal spot has no effect on resolution, high detail screens will yield better resolution than high speed screens.

In practice, however, neither the focal spot is infinitely small nor are the structures of interest in intimate contact with the film-screen combination. Under these circumstances, resolution depends on the MTF of the focal spot as well as on that of the screens.

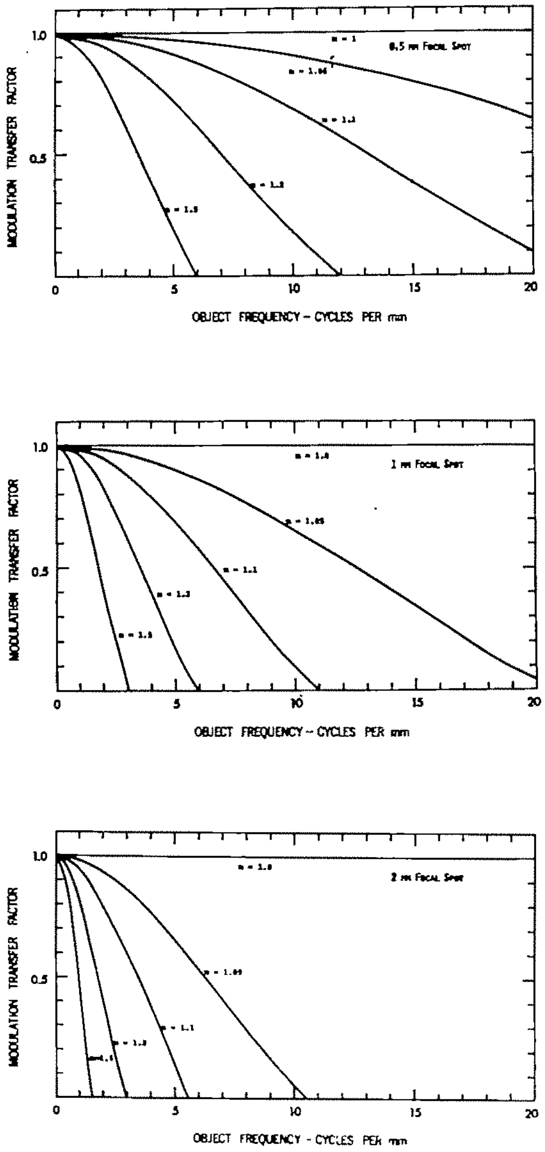

Fig. 2. The modulation transfer functions of focal spots of various size: *m* is the magnification factor, defined by the ratio $m=(d_1+d_2)/d_1$, where d_1 is the focus-object distance and d_2 is the object-film distance.

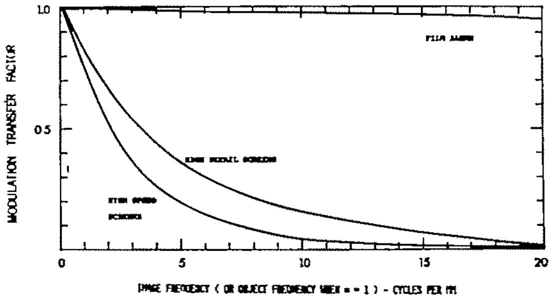

Fig. 3. The modulation transfer functions of 3 typical imaging systems, plotted against spatial frequency in the image plane.

The MTF of a focal spot is a strong function of the magnification ratio *m*, defined by the equation

$$m = \frac{d_1 + d_2}{d_1}, \qquad (1)$$

where d_1 is the focus-object distance and d_2 is the object-film distance. The relationship between focal spot size, magnification and focal spot MTF may be expressed by the equation

$$\text{MTF} = \frac{\sin \pi f_0 a(m-1)/m}{\pi f_0 a(m-1)/m}. \qquad (2)$$

This is the well known equation of Morgan,[1] suitably modified and expressed in terms of the effective focal spot size *a*, magnification factor *m* and object spatial frequency f_0.

THE MTF OF X-RAY FOCAL SPOTS

Figure 2 shows the modulation transfer functions of focal spots of various size at various magnification factors, calculated from Equation 2 and plotted in terms of spatial frequency in the object plane. Notice how rapidly the MTF deteriorates with the focal spot size and magnification factor *m*.

THE MTF OF SCREENS

Figure 3 shows the basic modulation transfer functions of a typical non-screen

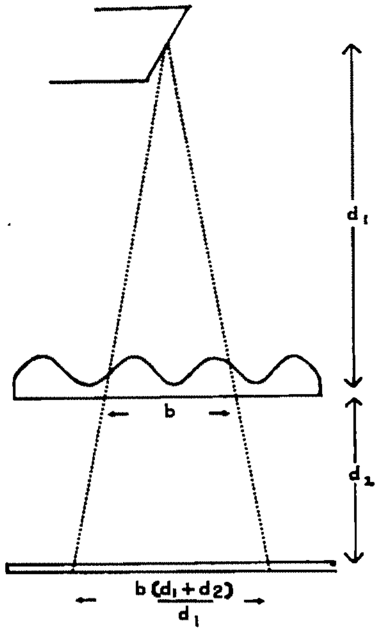

Fig. 4. Diagram illustrating the relationship between spatial frequency in the image plane and spatial frequency in the object plane. Because of magnification, a length b in the object plane becomes equal to a length $b(d_1+d_2)/d_1$ or bm in the image plane. Therefore, a spatial frequency of f_0 in the object plane becomes equal to $1/m$ times f_0 in the image plane. In other words, $f_0=mf_i$.

film, a screen type film exposed with high detail screens and a screen type film exposed with high speed screens, all plotted as functions of spatial frequency in the image plane. The experimental procedure used in generating these curves has been described elsewhere.[3] Now, because of magnification, a spatial frequency of f_0 in the object plane becomes equal to f_0/m in the image plane. In other words, the image frequency f_i and the object frequency f_0 are related to each other through the equation $f_0=mf_i$ (Fig. 4). Therefore, if we plot the screen MTF in terms of the object frequency, it follows that each of the curves shown in Figure 3 will give rise to a family of MTF curves corresponding to different values of the magnification factor m. Figure 5 shows the modulation transfer functions of the 3 imaging systems, now plotted against spatial frequency measured in the object plane. Notice how in each case, resolution improves progressively with the magnification ratio m.

We thus see that the MTF of a film or film-screen combination expressed in terms of spatial frequency in the object plane progressively improves with the magnification ratio, while the MTF of a focal spot progressively deteriorates with the magnification ratio.

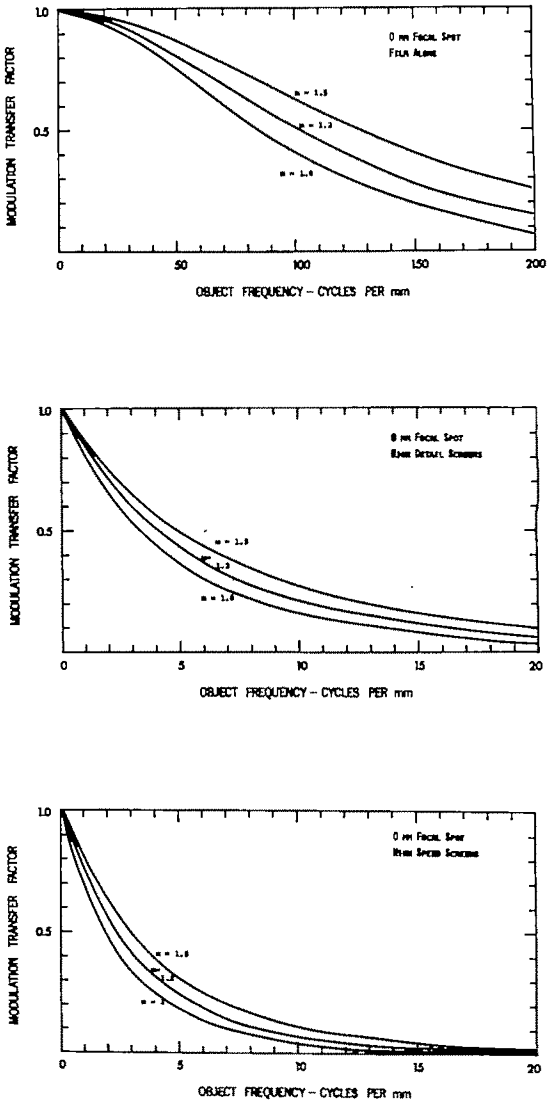

Fig. 5. The modulation transfer functions of Figure 3, replotted in terms of spatial frequency in the object plane.

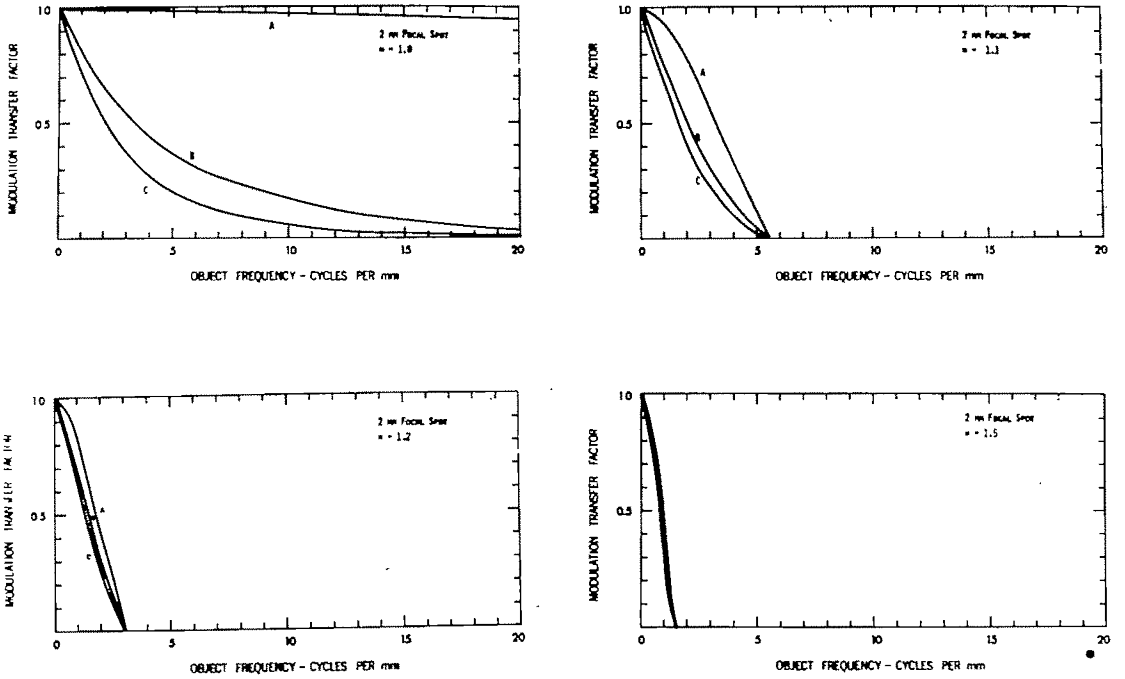

Fig. 6. The composite modulation transfer functions of a non-screen film (Curve A), high detail screens (Curve B) and high speed screens (Curve C) when used with a 2 mm. focal spot.

THE COMPOSITE MODULATION TRANSFER FUNCTIONS OF TYPICAL IMAGING SYSTEMS

Figure 6 shows the composite modulation transfer functions of the 3 imaging systems when used with a 2 mm. focal spot. Each of these curves was generated by multiplying together the modulation transfer factor of a 2 mm. focal spot with that of the imaging device in question at each of several points on the spatial frequency scale. Notice that the superiority of high detail screens over high speed screens diminishes with magnification. Indeed, the resolution characteristics of a 2 mm. focal spot are so poor that even at a magnification factor of 1.1, the difference between high speed screens and high detail screens virtually vanishes.

Figure 7 shows similar curves drawn for a 1 mm. focal spot. These curves suggest that with this focal spot, high detail screens may be expected to yield better resolution than high speed screens up to a magnification factor of 1.2.

Figure 8 shows the data for a 0.5 mm. focal spot. Here the superiority of detail screens is retained up to a magnification factor of 1.5.

In general, the smaller the focal spot size, the greater is the benefit of high detail screens over high speed screens from a resolution standpoint.

EXPERIMENTAL CONFIRMATION

We have tested the validity of the above conclusions experimentally by making radiographs of a large number of test patterns and phantoms. However, since the differences between high detail screens and high speed screens are rather small even at unit magnification, it has not been possible to demonstrate the results of these experiments by means of photographic reproductions; one has to look at the original radiographs themselves.*

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* Films can be made available to interested individuals on request.

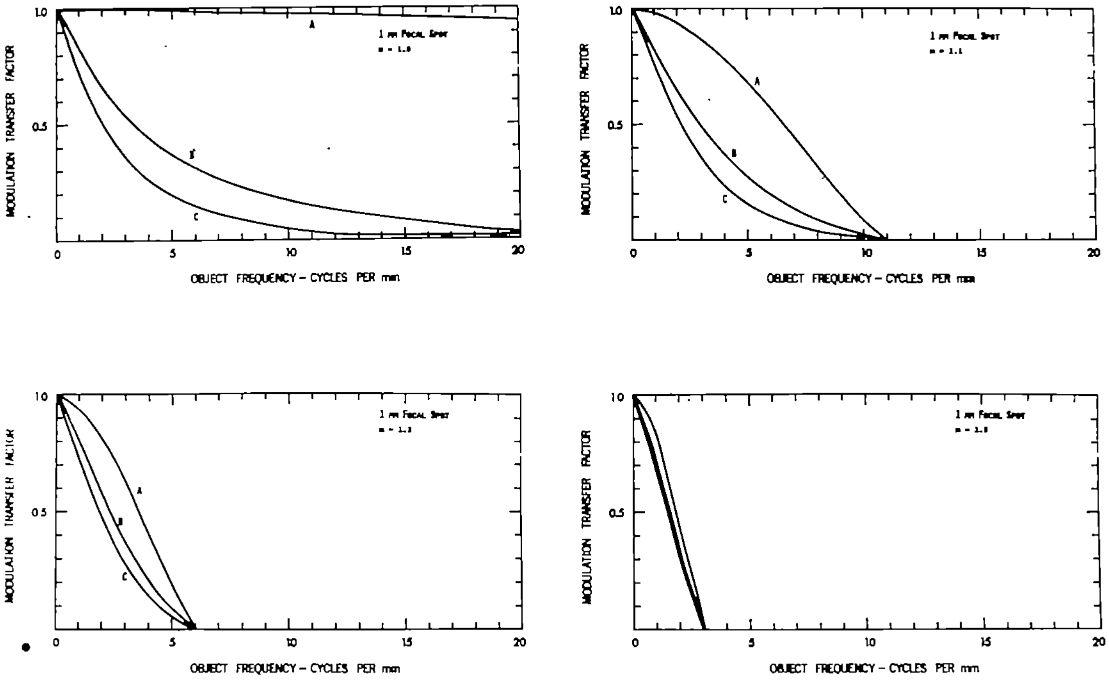

Fig. 7. The composite modulation transfer functions of a non-screen film (Curve A), high detail screens (Curve B) and high speed screens (Curve C) when used with a 1 mm. focal spot.

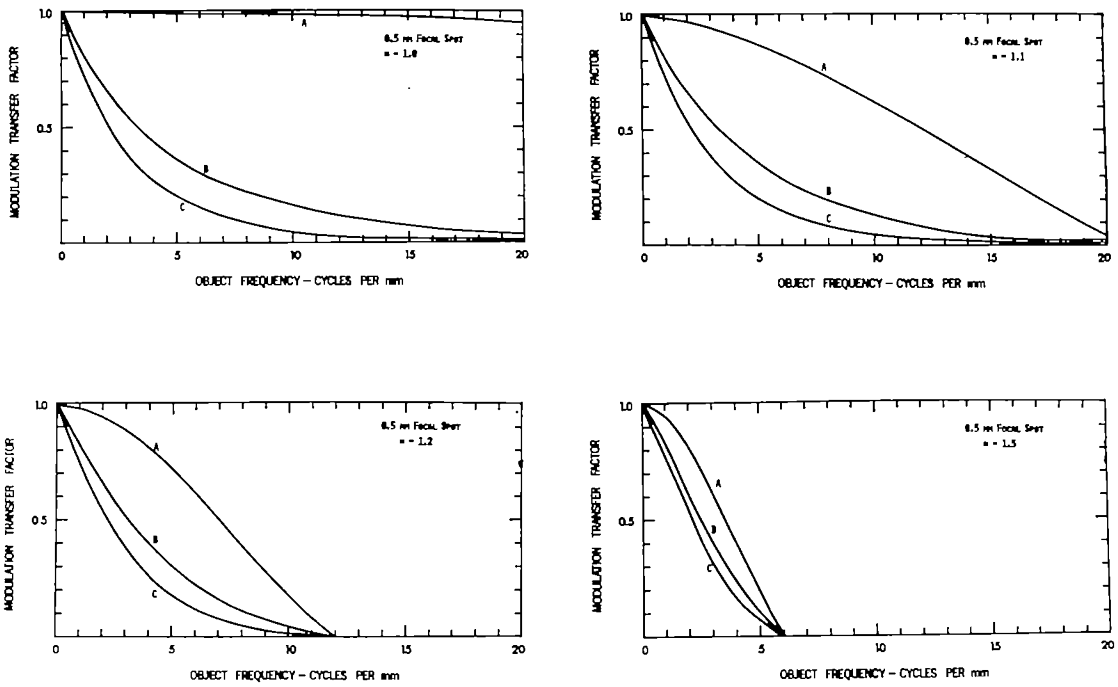

Fig. 8. The composite modulation transfer functions of a non-screen film (Curve A), high detail screens (Curve B) and high speed screens (Curve C) when used with a 0.5 mm. focal spot.

TABLE I

AVERAGE MAGNIFICATION FACTORS FOR TYPICAL BODY THICKNESSES*

Body Thickness (cm.)	Magnification Factor at		
	Film-side Skin Surface	Mid-Position	Tube-side Skin Surface
10	1.05	1.11	1.18
20	1.05	1.18	1.33
30	1.05	1.25	1.54
40	1.05	1.33	1.82

* Assumes a focus to film distance of 100 cm. and a table-top to film distance of 5 cm.

DISCUSSION

Detail screens undoubtedly yield better resolution than high speed screens if the objects being imaged are stationary and are in intimate contact with the film-screen combination. However, the improvement observed in many types of radiographic practice is often vanishingly small. This is because of the combined influence of screens, anatomic motion and focal spot size on radiographic resolution. The influence of each of these factors depends to a considerable extent on the magnification involved in the imaging system.

Table 1 shows the approximate magnification factors involved in a typical radiographic examination. From this table, it is clear that except in the case of structures located close to the film, detail screens will not give better resolution than high speed screens, if a 2 mm. focal spot is used. With a 1 mm. focal spot, theoretically some improvement in resolution may be expected up to a magnification factor of 1.2. In practice, however, even this is hard to realize for the following reasons: (a) it is not uncommon to find that the size of a so-called 1 mm. x-ray focal spot is in reality substantially larger than 1 mm.[3]; (b) the size and shape of an x-ray focal spot vary considerably with the angle of projection and hence a 1 mm. focal spot along the central axis of the beam is substantially larger toward the cathode end of the tube; and (c) a radiographic image is a superimposition of the shadows of a number of structures located at varying distances from the film.

CONCLUSION

In the foregoing discussion, it has been assumed that the subjects radiographed are stationary. If there is object motion, high detail screens will exhibit additional unsharpness because of the larger exposure times needed. This will further diminish the difference in resolution between the two types of screen. Indeed, if the relative speed of the 2 types of screens is widely different and the object motion is quite large (say greater than 1 or 2 mm. per sec.), high speed screens (because of the reduced time of exposure) may yield better resolution than detail screens.

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The author is indebted to Dr. Russell H. Morgan for his valuable guidance throughout the progress of this work. Thanks are also due to Mrs. Sandra Mervis for her secretarial assistance.

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3. RAO, G. U. V., and BATES, L. M. Modulation transfer functions of x-ray focal spots. *Physics in Med. & Biol.*, 1969, *14*, 93–106.

AUGUST, 1971

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Representative on the Board of Chancellors of the American College of Radiology: Juan A. del Regato.

Fifty-fourth Annual Meeting: Boca Raton Hotel and Club, Boca Raton, Florida, May 14–19, 1972.

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* Deceased.

EDITORIALS

INSTRUCTION COURSES OF THE SEVENTY-SECOND ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

A TRUE EXPRESSION OF "INTEGRATED RADIOLOGY"

PRESIDENT-ELECT Thomas M. Fullenlove, Chairman of the Program Committee, requested Dr. Harold O. Peterson, Director of the Section on Instruction, to again arrange Instruction Courses for the Seventy-second Annual Meeting of the American Roentgen Ray Society, which will be held at the Sheraton-Boston Hotel, Boston, Massachusetts, September 28-October 1, 1971.

These courses, inaugurated 32 years ago by Dr. Edward L. Jenkinson, President-Elect of the Fortieth Annual Meeting of the Society in Chicago, Illinois, since then amply reflected the Society's interest in "integrated radiology"[1] by bringing year after year, to members and their guests the most authoritative information about the latest developments in all branches of radiology and its allied sciences.

Exactly 2 decades ago, at the Fifty-second Annual Meeting of the Society held in Washington, D.C., inspired by the same theme of "integrated radiology," at the initiative of Lawrence Reynolds,[2] world-renowned Editor of this Journal, the term of "Nuclear Medicine" was introduced, and to connote the Society's interest in this newest branch of radiology, the title of the official organ was changed from The American Journal of Roentgenology and Radium Therapy to The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine. This historic connotation had great impact on the world radiologic literature and the term "Nuclear Medicine" has now become commonplace.

Another step toward "integrated radiology" was undertaken by President-Elect James C. Cook,[3] when at the Seventieth Annual Meeting of the Society, held in Washington, D.C., in 1969, he introduced the innovation of inviting each National Subspecialty Society in Radiology to select from the papers read at its Annual Meeting one, which is of "greatest interest to all radiologists," for presentation as an "Award Paper" at the Annual Meeting of the American Roentgen Ray Society. This procedure has also been successfully followed by President-Elect John F. Roach[4] at the Seventy-first Annual Meeting, and is now being followed by President-Elect Tom M. Fullenlove at the Seventy-second Annual Meeting.

The Society is extremely fortunate to have again Dr. Harold O. Peterson as the brilliant Director of the Section on Instruction, who now for a decade and a half has embodied the principle of "integrated radiology" in selecting outstanding Faculties of Instruction, dealing with subjects covering all branches of radiology and its allied sciences. This year's Courses are the most numerous and—judging from a perusal of the titles and abstracts on the following pages—the most excellent in the Society's history.

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[1] Leucutia, T. Integrated Radiology—President's Address, 1961. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1961, *86*, 803–806.

[2] Scientific Proceedings and Minutes of the Fifty-second Annual Meeting, American Roentgen Ray Society, Shoreham Hotel, Washington, D. C., September 25–28, 1951, p. 64–65; 86–87.

[3] Cook, J. C., President-Elect, 1969. The Seventieth Annual Meeting of The American Roentgen Ray Society. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, p. 657.

[4] Roach, J. F., President-Elect, 1970. The Seventy-first Annual Meeting of The American Roentgen Ray Society. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, p. 637.

The Section of Instruction will present a total of 59 courses: 16 each on Tuesday, Wednesday and Thursday; and 11 on Friday. Some of the courses will be in 2 sessions, and 1 course in 3 sessions. Dr. W. B. Young, London, England, this year's distinguished Caldwell Lecturer, will give a most interesting course on "Primary Malignant Tumors of Bone" (Course 312) on Thursday.

The distribution of the courses according to categories is as follows: clinical diagnostic radiology, 21; pediatric radiology, 5; neuroradiology, 6; cardiovascular radiology, 5; radiologic-pathologic correlation, 4; therapeutic radiology, 6; nuclear medicine, 5; technology, 3; and miscellaneous, 4. Some of the courses represent "integrated" interest in 2 or even 3 categories, as for instance, Course 113, titled "Hydrocephalus—Present Status of Radiological and Radionuclide Investigation," which incorporates procedures pertaining to the domain of neuroradiology, pediatric radiology and nuclear medicine.

The rooms in which the Courses will be held are all provided with ultramodern facilities and all are air-conditioned. The respective assignments will be posted at the Special Registration Desk.

A list of the distinguished Faculty of Instruction, the title and a brief abstract of each course, as well as the condensed schedule of the courses and the registration form with the appropriate instructions (Pink Sheet) are published in the following pages of this issue of the JOURNAL.

Director Peterson recommends a registration as early as possible.

T. LEUCUTIA, M.D.

[redacted]

General Hospital) to become the Director of Radiology.

Last year at the age of 65 years, he retired from the position of Director and was appointed Emeritus Professor of Radiology at Case Western Reserve University. A recognition dinner in honor of his retirement was held and many of his more than 90 former residents traveled across the country and from Canada to attend. He was to have been honored at the 1971 meeting of the Ohio State Radiological Society.

Dr. Hauser had been Director of Radiology at University Hospitals of Cleveland from 1942–1946 and was Director of Radiology at Grace Hospital in Cleveland at the time of his demise. He held consultant or honorary appointments to Sunny Acres Sanitorium, Forest City Hospital, Marymount Hospital, Cleveland State Hospital, Lutheran Hospital and Lakewood Hospital.

Dr. Hauser had numerous publications to his credit and he was an illustrious Associate Editor of the American Journal of Roentgenology, Radium Therapy and Nuclear Medicine, and Chairman of the Publication Committee of the American Radium Society for more than 2 decades, past President and Trustee of the Cleveland Medical Library Association. He was a member of numerous professional societies and a past President of the Cleveland Radiological Society. His advice in matters of medical ethics was highly valued.

Dr. Hauser was always a considerate person and conscious of his obligations to his fellow men. He was a Charter Member, and a member of the Board, of the Temple Emanu El.

He is survived by his wife, 2 sons and 7 grandchildren. His wise and gentle guidance as a friend, counsellor and physician will be deeply missed.

Alvin Segel, M.D.

3345 Scranton Road
Cleveland, Ohio 44109

* * *

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

Harold O. Peterson, M.D., *Director*

Titles and Abstracts of Courses Offered
Seventy-Second Annual Meeting
The Sheraton-Boston Hotel, Boston, Massachusetts
September 28–October 1, 1971

The Instruction Courses for the Seventy-second Annual Meeting of the American Roentgen Ray Society will be held on Tuesday, Wednesday and Thursday afternoons, September 28, 29, and 30, at 3:00 P.M., and Friday afternoon, October 1, at 1:30 P.M. Each course will be of 90 minutes' duration and no other official activity will be scheduled for this time.

A special registration desk for these courses will be located near the general registration area. Complete information concerning the courses as well as tickets for the courses will be available on Monday, September 27 from 8:30 A.M. to 4:00 P.M. and daily the rest of the week.

Sixteen daily courses will be given on Tuesday, Wednesday and Thursday, and eleven on Friday. Please note that some of the courses will be given in two sessions. A brief abstract for each course will be found on the following pages as well as a complete list of the faculty and the registration form.

Admission to all courses will be by ticket. To avoid the confusion usually present on the first day, tickets will be mailed to everyone whose registration forms (Pink Sheets) reach the office of the Director before September 17, 1971. Any orders received after this date will be filled and the tickets will be held at the Registration desk. Please watch for your tickets which will be sent in an envelope marked: "Section on Instruction—American Roentgen Ray Society—Tickets Enclosed."

All orders will be filled according to the postmark on the envelopes and the first mailing will be about September 7, 1971.

HOW TO REGISTER AND OBTAIN TICKETS

Review the abstracts on the following pages; make three choices for each day; fill out the pink order sheet with the course number and name of instructor for *each choice*. For each course selected a $3.00 registration fee is required from nonmembers. Four tickets, one for each day, may be purchased for $10.00, but single tickets are $3.00 each.

Members of the American Roentgen Ray Society, graduate students or residents in Radiology, and nonmembers who are contributing to the program either by way of an instruction course, a paper or scientific exhibit, are not required to pay for these courses but must fill out a Pink Order Sheet indicating their choices.

If your advance registration reaches the office of the Director after September 17, 1971, you may obtain your tickets at the Registration Desk of the Section on Instruction at the hotel on Monday afternoon, September 27, 1971, and thereafter.

Please mail your order sheets to Dr. Harold O. Peterson, 1995 West County Road "B", St. Paul, Minnesota 55113.

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W. B. Young, M.D., Director of Radiology, The Royal Free Hospital, London W.C.1, England

DESCRIPTION OF COURSES

COURSE 101
and
COURSE 201

Tuesday and Wednesday

HAROLD G. JACOBSON, M.D.
Bronx, New York

Course 101. Benign Bone Tumors: Interesting Manifestations

A large number of cases will be presented to illustrate the characteristic radiologic features of the various benign bone tumors, both commonplace and rare.

Among the important considerations to be stressed will be: the use of the zone of transition in distinguishing a benign from a malignant lesion; the diagnostic characteristics of different patterns of periosteal reaction; evaluation of the tumor matrix; analysis of the soft tissue component of an expanding lesion; and location of the tumor with emphasis on the predilection of different neoplasms for specific skeletal areas.

The material will include the following examples of benign skeletal neoplasms: some commonly encountered tumors; curious and exotic neoplasms; tumors in unusual locations; and aggressive benign lesions which may simulate malignant neoplasms.

Course 201. Some Interesting Features of Skeletal Radiology: Challenges in Diagnosis

A good part of the intellectual stimulation inherent in diagnostic radiology rests in the challenge afforded by the opportunity of viewing the films of esoteric conditions and the unusual manifestations of more common abnormalities.

In this course a number of such unusual skeletal disorders will be presented in groups. Diagnostic challenges will be provided by the introductory cases in each area, which will also serve as a point of departure for the presentation of other appropriate examples of a particular entity. The importance of a careful analytic evaluation of the fundamental radiologic features of each disorder will be stressed, demonstrating that even in the most challenging of cases the film speaks its specific language, which we must learn to understand.

COURSE 102
COURSE 202
and
COURSE 302

Tuesday, Wednesday and Thursday

A. S. MACMILLAN, JR., M.D.
Boston, Massachusetts

Course 102. Sinuses and Orbits

Following a review of the basic projections using table-top techniques, there will be a discussion of anteroposterior and lateral tomography of the facial region. Comments will be made on orthopantomography, sialography, and dacryocystography, intraorbital foreign body localization, orbital venography, radioactive isotopes, positive contrast orbitography, carotid arteriography, and ultrasound as related to the orbit.

Course 202. Temporal Bone

This will begin with plain film roentgenograms which are still the scout roentgenograms of the area. Lesions best seen in each view will be shown. Tomography will be included. Pantopaque cisternography will be discussed plus the newest clinical testing in acoustic nerve tumor detection.

Course 302. Larynx, Pharynx, Cervical Esophagus and Trachea

The everyday approach to the airway and the pharynx and cervical esophagus will be presented using plain film roentgenograms, fluoroscopy, and spot roentgenograms, tomography, supervoltage exposure and positive contrast with emphasis on tantalum powder.

COURSE 103
and
COURSE 203

Tuesday and Wednesday

SIDNEY W. NELSON, M.D.
Columbus, Ohio

Course 103. The Discovery of Gastric Ulcers and the Differential Diagnosis Between Benignancy and Malignancy

The discovery of gastric ulcers and the differential diagnosis between benignancy and malignancy are common problems which often present difficulties. After a brief description of the author's technique of examining the stomach and duodenum, the following differential diagnostic signs will be discussed and demonstrated:

1. *Signs of benignancy*
   A. The penetrating sign
   B. Mucosal folds radiating to the edge of the crater
   C. Hampton's line
   D. Ulcer collar
   E. Ulcer mound
   F. Blood clot in floor of crater
   G. Concavo-convex ("quarter moon") shaped barium collection
2. *Signs of malignancy*
   A. Carman's meniscus sign
   B. Kirklin's meniscus complex
   C. The abrupt transition sign

   - D. Nodularity of the tissue surrounding the crater
3. *Nonspecific criteria*
   - A. Intraluminal location of the crater
   - B. The "ring shadow"
   - C. Size of the crater
   - D. Location of the crater
   - E. Shape of the crater
   - F. Change of size of the crater during treatment

Those craters which do not clearly show reliable signs of benignancy or malignancy should be termed "indeterminate." The proper spot roentgenograms of gastric ulcers, utilizing maneuvers designed to bring out the above mentioned diagnostic signs of benignancy and malignancy with techniques which *profile* the crater, will result in the highest rate of diagnostic accuracy.

Course 203. Fundamentals in the Roentgenologic Study of Skeletal Trauma

The roentgen study of injured patients forms a significant part of the every day workload of most general diagnostic radiologists. The inability of patients to cooperate during the radiologic examinations, the use of mediocre radiologic equipment, the analysis of roentgen findings by relatively inexperienced physicians, and the urgency of making decisions relative to treatment, all serve to create situations in which subtle, but important, abnormalities may not be diagnosed. Furthermore, necessary additional studies may not be obtained if the physicians studying the roentgenograms do not recognize abnormalities or lack knowledge about the mechanisms of injury with resultant failure to obtain additional studies which are indicated. In an attempt to help younger radiologists become better prepared to analyze roentgen findings and perform the best roentgen studies of injured patients, this course will emphasize the following:

1. *Technical principles which are often of importance in making the diagnosis*
2. *The importance of understanding the mechanisms of injury in such lesions as*
   - A. Monteggia fracture dislocation
   - B. Galeazzi fracture dislocation
   - C. Tibio-fibular diastasis
   - D. Metaphyseal "corner fractures" in children
   - E. "Chip fractures" of the anterior superior corner of a cervical vertebral centrum
   - F. Anterior and posterior hip dislocations
   - G. Compression fracture of the lumbar spine associated with fractured calcaneus
   - H. "Chance fracture" of the lumbar vertebrae associated with seat belt injuries
3. *The importance of remembering normal anatomic relationships in diagnosing the following abnormalities*
   - A. Lunate dislocations
   - B. Dislocation of the radial head
   - C. Epicondylar fracture of the radius
   - D. Fracture dislocation of the epiphysis of the medial epicondyle of the humerus
4. *Some of the subtle, but important signs of trauma*
   - A. "Fat pad" sign in elbow injuries
   - B. The "pronator quadratus fat-pad sign" in wrist injuries
   - C. The double white articular cortical line
   - D. Fat-fluid levels in large joints due to fractures which involve articular surfaces
   - E. Traumatic pneumocephalus
   - F. Air in pelvic soft tissues following pelvic trauma

COURSE 104
and
COURSE 204

Tuesday and Wednesday

E. R. N. GRIGG, M.D.
Oak Park, Illinois

Course 104. The History of Radiology

I. *Its First Four Periods*

1. The Era of the Roentgen Pioneers
2. The Golden Age of Radiology
3. The Atomic Phase
4. The Computer Stage

By and large, the transition from the Era of the Pioneers into the Golden Age of Radiology occurred during World War I, while World War II separated the Golden Age from the Atomic Phase. Not only did the first two World Wars coincide with, they also contributed to, the respective transition into another period in the history of radiology. The Computer Stage started around 1965.

Lantern slides will be used in rekindling the memory of those men who with their ideas and impetus precipitated the transition from each period to the next. Historic reviews (hindsight) tend to improve one's understanding of the present, and occasionally allow an extrapolated glimpse into the future (foresight).

Course 204. The History of Radiology

II. *Radiologic Organizations*

Professional associations, whether economic (tradesmanship) or scientific (craftsmanship) or mixed, grow only if the aims of their founders coincide with the aims of potential members, and survive only so long as the initial aims are being altered in response to changing conditions.

The very first American radiologic organization, the ARRS, was founded by the pioneers in 1900. The RSNA appeared in 1915 as the Golden Age of Radiology approached. In 1940 today's ACR (in reality the Inter-Society Committee using an "old" name) heralded the coming of the Atomic Phase. Our involvement in certain paramedical societies, such as the Association of Physicists in Medicine and the American Society of Radiologic Technologists, reflects the participation of physicists and technologists as indispensable members of the radiologic team.

Interest in, and a working knowledge of, the history of radiologic organizations will prove to be very helpful to all those who are or intend to become officers of any such regional or national group.

COURSE 105
and
COURSE 205

Tuesday and Wednesday

ROBERT W. MCCONNELL, M.D.
Dallas, Texas

JOHN W. LANE, M.D.
Little Rock, Arkansas

Nuclear Medicine

• This two day course is designed to orient the general radiologist in the field of nuclear medicine, with emphasis on scanning procedures. It will serve both as an introduction to those who wish to enter the field, as well as a refresher for those already performing these studies in their radiologic practice.

Course 105. The first day's discussion will cover

1. Brief review of basic physics
2. Radiation protection
3. Radio-pharmaceuticals
4. Instrumentation and techniques

Course 205. The second day's discussion will cover

5. Interpretation and correlation with associated radiographic studies:
   a. Dynamic studies
   b. Static scans
6. Economics

While it would appear that the second day's discussion logically follows the first, the sessions are so constructed that they are not mutually dependent. Therefore, those who wish may attend either one separately.

COURSE 106
and
COURSE 306

Tuesday and Thursday

E. ROBERT HEITZMAN, M.D.
Syracuse, New York

Course 106. Roentgen Pathologic Correlations in Common Pulmonary Disorders

An analytic and deductive approach to the diagnosis of abnormalities demonstrated on roentgenograms requires a thorough knowledge of the gross pathologic appearances of these abnormalities. Although this is vitally important in pulmonary radiology, the pathology of many pulmonary diseases is often poorly understood. Roentgen pathologic correlations will be demonstrated to establish a firm pathologic basis for the roentgen appearances in common pulmonary conditions such as pulmonary infarct, pneumonia, emphysema, pulmonary edema, and some chronic diseases of the pulmonary interstitium.

Course 306. The Mediastinum: Roentgen Anatomic Correlations

Although the anatomy of the mediastinum is complex, many mediastinal structures can be evaluated by careful radiographic analysis of the interfaces of the mediastinum and lung. The normal roentgen anatomy of the right and left paratracheal areas, the azygous vein, the azygoesophageal recess and the prespinal and paraspinal lines will be discussed. Examples will be shown to emphasize the importance of these anatomic concepts to the evaluation of mediastinal and pulmonary disease.

COURSE 107

Tuesday

JOHN G. MCAFEE, M.D.
Syracuse, New York

Current Status of Radioisotopic Methods in the Diagnosis of Renal Disease

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COURSE 108

Tuesday

ANTHONY F. LALLI, M.D.
Cleveland, Ohio

The Tailored Urogram

A discussion of the various types of urograms and the methods of approach to use in various clinical problems will form the main element of this course. The approach to the hypertensive patient is quite different from that of the patient suffering from ureteral colic and the differences in these as in other problems will be illustrated.

The patient who is identified as having a renal mass on urography presents a special problem which can be quickly resolved on an out-patient basis by doing a guided percutaneous puncture. The indications and methods of this examination will be dealt with in detail, as will be the performance of the percutaneous antegrade pyelography in patients with duplication anomalies and obstructive uropathy.

A portion of the time will be spent delineating the problems of pyelonephritis, renal papillary necrosis, medullary sponge kidney disease, and the interrelationships and importance of these problems.

COURSE 109

Tuesday

HARVEY I. MEYERS, M.D.
Los Angeles, California

Intestinal Obstruction and the Detection of Small Amounts of Intraperitoneal Fluid

The course will deal with the development of a systematic approach to intestinal obstruction, including the analysis of plain film roentgenograms of the abdomen and the use of contrast media in intestinal obstruction.

The last part of the discussion will deal with the detection of small amounts of intraperitoneal fluid and its importance in the diagnosis of acute abdominal conditions.

COURSE 110

Tuesday

CHARLES E. SHOPFNER, M.D.
Kansas City, Missouri

Current Status of Pediatric Uroradiology

When this course was first presented several years ago, it was necessary to spend a great deal of time presenting iconoclastic material proving that urinary tract infection is not always secondary to obstruction and anomaly, that most hydronephrosis is nonobstructive, and that reflux is nonobstructive.

It is now possible to present positive information based on sound clinical investigation and study of such problems as reflux, pathology of the refluxing ureter, the primary effects of infection on the urinary tract and other important problems in pediatric genitourinary radiology.

COURSE 111

Tuesday

EDWARD B. SINGLETON, M.D.
Houston, Texas

Pediatric Pulmonary Abnormalities

Because the pediatric patient may be afflicted with many of the lung diseases encountered in the adult patient as well as a large number of specific pulmonary problems found only in infants and children, a complete discussion of this subject is impossible. Therefore, the course will primarily consider causes of respiratory distress in the newborn and young infant. This age group is important not only to the obstetrician, pediatrician and pediatric radiologist, but also to the general radiologist in his contact with the hospital maternity and newborn sections.

The causes of respiratory distress in this young age group are numerous and include extrapulmonary conditions such as congenital heart disease, intestinal obstruction, diaphragmatic hernia and central nervous system damage.

The conditions which will be discussed include the following:

Primary pulmonary diseases producing respiratory distress in the newborn and young infant
- Transient tachypnea of the newborn (wet lung syndrome)
- Respiratory distress syndrome
- Aspiration syndrome
- Pulmonary hemorrhage
- Intrauterine infectious pneumonia
- Aspiration pneumonitis
- Wilson-Mikity syndrome
- Bronchopulmonary dysplasia
- Pulmonary lymphangiectasia
- Pulmonary agenesis

Surgical or mechanical abnormalities producing respiratory distress in the newborn and young infant
- Obstruction of upper air passages
- Esophageal atresia and tracheoesophageal fistula
- Pneumomediastinum and pneumothorax
- Congenital lobar emphysema
- Congenital cystic adenomatoid malformation
- Phrenic nerve paralysis and eventration
- Diaphragmatic hernia
- Accessory diaphragm
- Vascular ring
- Chylothorax
- Congenital deformities of the thorax

If time permits some of the common and rare pulmonary infections encountered in the young infant will be included.

COURSE 112

Tuesday

HOWARD J. BARNHARD, M.D.
Little Rock, Arkansas

The Use of Computers in Diagnostic Radiology

Digital computers are still relatively uncommon in medicine. As such, they largely represent an unknown quantity to most of us and inspire a range of emotions from awe to antagonism. However, they have the potential for serving mankind as a very useful device, and should be thought of as an extension of our capabilities just as the desk calculator improved our ability to handle addition and subtraction.

A variety of current and potential applications in diagnostic radiology will be covered including the following:

Operations, such as patient scheduling

Record keeping of both departmental and patient data
Auto-analysis of the diagnostic report for coding
Assistance in interpretation ranging from image enhancement to computer-aided diagnosis

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COURSE 113

Tuesday

PAUL F. J. NEW, M.D.
Boston, Massachusetts

ROGER A. BAUMAN, M.D.
Boston, Massachusetts

HENRY P. PENDERGRASS, M.D.
Boston, Massachusetts

Hydrocephalus—Present Status of Radiological and Radionuclide Investigation

This course will consist of a review of the clinical, anatomic and pathophysiologic elements of hydrocephalus. The value and limitations of plain film roentgenographic examination of the skull, cerebral angiography and cerebral pneumography will be subjected to critical review, complemented by presentation of illustrative cases of hydrocephalus due to a variety of pathologic entities.

A systematic classification appropriate to present knowledge of pathophysiology, and to present methods of investigation, will be offered.

The techniques of radionuclide investigation of cerebrospinal fluid formation, movement and absorption will be described in some detail, followed by an analysis of normal and pathologic varieties of radionuclide scans.

The presentation will emphasize correlations between scans, conventional radiologic studies and the results of ventricular shunting.

COURSE 114

Tuesday

WILLIAM T. MESZAROS, M.D.
Chicago, Illinois

The Pulmonary Circulation and Pulmonary Vessels

Accurate interpretation of chest roentgenograms depends on a thorough knowledge of the roentgen manifestations of diseases which affect the pulmonary circulation and pulmonary vessels. Plain film roentgenographic and angiocardiographic findings will be presented in the following conditions:

- The Pulmonary Circulation
  - Decreased flow
  - Increased flow
  - Increased resistance
  - Pulmonary venous hypertension
  - Combined arterial and venous hypertension
- Diseases of the Pulmonary Vessels
  - Small pulmonary arteries
  - Pulmonary artery enlargement
  - Stenosis of the pulmonary artery and its branches
  - Absence of a pulmonary artery
  - Aberrant left pulmonary artery
  - Varicosities of the pulmonary veins
  - Partial anomalous pulmonary venous connection
  - Total anomalous pulmonary venous connection
  - Cor triatriatum
  - Pulmonary arteriovenous fistula
  - Pulmonary sequestration

COURSE 115

Tuesday

ROBERT E. WISE, M.D.
Boston, Massachusetts

Current Concepts of Cholangiography

The course will cover all of the commonly used types of direct and indirect approaches to the examination of the biliary tract.

This will include intravenous cholangiography, percutaneous transhepatic cholangiography, postoperative T-tube cholangiography, operative cholangiography and barium cholangiography.

Indications, technique, complications, and diagnostic criteria for each modality will be discussed.

COURSE 116

Tuesday

MORTON M. KLIGERMAN, M.D.
New Haven, Connecticut

Tricks and Treatment Planning

Appreciation of the increasing curability of tumors by high doses of irradiation and the current availability of high energy, high output, highly accurate x-ray generators has increased the need for the meticulous application of the radiation energy to the patient.

Careful technique must be observed in making contours, obtaining appropriate port films, and in the application of abutting fields.

The limitation of certain types of equipment in accomplishing localized high dose therapy with minimal risk of untoward effect will be discussed.

COURSE 206

Wednesday

JOHN L. GWINN, M.D.
Los Angeles, California

Roentgen Manifestations of Acquired Bone Disease in Children

Many of the acquired bone lesions seen in children are of the same nature as in the adult. However, due to the growing bone, the roentgen changes may be different. Also, some acquired lesions are primarily seen in the pediatric age group, and longitudinal studies are of great value.

We will illustrate the changes in:

1. Deficiency states
2. Trauma (including the "battered child")
3. Osteochondritis
4. Bone and joint infection
5. Intoxication with various substances
6. Blood dyscrasias
   a. Leukemia
   b. Sickle cell disease
   c. Cooley's anemia
   d. Hemophilia
7. Metabolic disease
8. Selected tumors
9. Miscellaneous

COURSE 207
and
COURSE 307

Wednesday and Thursday

D. GORDON POTTS, M.D.
New York, New York

Course 207. Pneumoencephalography

1. Technique
   a. Filling of ventricles
   b. Manipulation of air into various cisterns
   c. Equipment for air studies—similarities and differences, with emphasis on tomography
2. Brief discussion of ventricular anatomy
3. Anatomy of the cisterns with emphasis on the ambient, cerebellopontine angle, and quadrigeminal plate cisterns. These cisterns will be demonstrated with the aid of brain specimens, plain film roentgenograms and tomograms

Course 307. Pneumoencephalography

1. Supratentorial masses
2. Infratentorial masses
3. Multiple masses
4. Deep masses and tumors involving the corpus callosum
5. Suprasellar masses
6. Tentorial masses
7. Cerebral atrophy
8. Information obtained from air studies that is not obtained from angiographic studies
9. Limitations and pitfalls of air studies

COURSE 208
and
COURSE 308

Wednesday and Thursday

J. S. DUNBAR, M.D.
Montreal, P. Q., Canada

Course 208. Pediatric Chest Radiology

Chronic diseases of the lung will be considered. The indications for special procedures such as bronchoscopy and angiography will be discussed, and examples of lesions for which special procedures are of diagnostic value will be presented.

The differential diagnosis of localized lesions and diffuse infiltrations of the lung will be outlined.

Course 308. Pediatric Radiology of the Gastrointestinal Tract

This course will cover a number of entities of the gastrointestinal tract of the infant and child which can be investigated by contrast media.

Emphasis will be placed on the relation of the planned investigation to the presenting complaints, and will include investigation of swallowing disorders, vomiting, abdominal pain, gastrointestinal bleeding, steatorrhea, constipation, and intestinal obstruction.

For each of these entities, the method of investigation most suitable to the presenting complaint will be outlined, followed by demonstration of conditions most likely to be encountered.

COURSE 209

Wednesday

MELVIN P. JUDKINS, M.D.
Loma Linda, California

Selective Coronary Arteriography

Coronary arteriography has become a *sine qua non* of the diagnosis and management of coronary obstructive heart disease. Coronary artery surgery is now technically feasible in a large number of patients who have life-threatening obstructive disease. If patients are carefully selected, postoperative improvement is immediate and dramatic.

High resolution serial and conventional cine angiographic techniques and isotope myocardial perfusion studies are used to evaluate coronary anatomy, ventricular dynamics, and myocardial viability.

In selecting patients for surgery, it is not enough to establish the presence of an obstructive lesion of the coronary artery; one must also evaluate accurately: (1) the degree of obstruction of the vessel; (2) the presence and extent of arteriosclerotic involvement of the vascular wall distal to the obstruction or occlusion, no matter how small the vessel may seem to be; (3) the potential run-off; (4) the defined perfusion deficit; (5) the potential for improvement; and (6) the therapeutic potential.

The course will concern itself with: (1) techniques of coronary arteriography; (2) the prevention of complications; (3) the evaluation of ventricular function; (4) the definition of perfusion deficit; and (5) the selection of patients for surgery.

COURSE 210
and
COURSE 310

Wednesday and Thursday

BENJAMIN FELSON, M.D.
Cincinnati, Ohio

Course 210. Diffuse Lung Lesions

The problem of differential diagnosis of the vast array of disseminated pulmonary lesions is an almost unsurmountable one unless specific patterns can be identified on the roentgenogram. It is often possible to recognize the diffuse alveolar pattern and, once identified, the list of its causes is relatively short. The recognition of diffuse interstitial disease is considerably more difficult, but even here it is sometimes possible to reliably distinguish this pattern and thereby reduce the etiologic possibilities to a reasonable number. In this course the roentgen features of the alveolar and interstitial patterns will be described and the various causes of each will be illustrated.

Course 310. Abdominal Gasmanship

COURSE 211

Wednesday

GUY D. POTTER, M.D.
New York, New York

Sialography

Methodology of obtaining sialograms: approach, patient comfort, contrast material, needles

Morphology: differences between the submaxillary gland and the parotid gland as reflected on the sialogram, measurement of duct, and the mean gland size

Normal variants: the sialographic appearance of normal variations of the anatomy of the salivary glands

Indications and contraindications for sialography

Differential diagnosis of swellings in the parotid gland and submaxillary areas

- Extraglandular causes of swelling
  - Clinical differential diagnosis
  - Sialographic differential diagnosis
- Glandular causes of swelling
  - Non-neoplastic
  - Neoplastic-clinical, histologic and sialographic differential diagnosis
    - Benign
    - Malignant
  - Autoimmune sialoses
    - Sjögren's syndrome
    - Mikulicz's disease
    - Recurrent parotitis in children
    - Recurrent parotitis in adults

COURSE 212

Wednesday

WILLIAM B. SEAMAN, M.D.
New York, New York

Radiology of the Larynx and Pharynx

The various techniques for the radiographic examination of the larynx and pharynx will be described and the indication for each will be discussed. These include contrast laryngography, plain film roentgenography of the neck, cine roentgenography using barium and tomography.

Examples of typical lesions will be presented. Particular emphasis will be placed on functional abnormalities of the pharynx and the mechanism of deglutition.

COURSE 213

Wednesday

WILLIAM J. TUDDENHAM, M.D.
Philadelphia, Pennsylvania

The Perception of the Roentgen Shadow: Its Mechanisms; Its Uncertainties; Its Control

The course will deal with current knowledge of the psychophysiology of perception as it relates to the identification of critical radiographic details. It will consider sources of perceptual error and various systems proposed for their control ranging from replicate reading to electronic image processing. Techniques for defining the logical analysis of roentgen images and the potential application of such techniques to the solution of perceptual problems will be emphasized.

COURSE 214

Wednesday

JOHN N. WOLFE, M.D.
Detroit, Michigan

Mammography

The course will be divided into the following sections:

1. Very brief historic review
2. Indications for mammography
3. Technique of examination

4. Radiographic aspects of the normal breast and benign, malignant and miscellaneous conditions
5. Results
6. Mammography as a screening examination for breast cancer
7. Newer techniques
    a. Molybdenum anode x-ray tubes
    b. Xeroradiography

Throughout the presentation slides utilizing conventional film mammograms or xeroradiographs will be used freely and randomly to present the particular diagnostic point.

A course outline will be furnished which will also contain a selected bibliography.

COURSE 215

Wednesday

ALEXANDER R. MARGULIS, M.D.
San Francisco, California

Radiology of Colonic Polyps

The course will deal with the significance of various types of colonic polyps, the radiologic approach to their detection and the syndrome of multiple polyps.

Various techniques and their advantages and disadvantages will be discussed.

Particular attention will be directed to the newest concepts of the relationship of polyps and cancer. Again, it appears that there is a more intimate relation between them than was fashionable to assume a few years ago.

COURSE 216

Wednesday

WILLIAM L. CALDWELL, M.D.
Nashville, Tennessee

Practical Palliative Radiotherapy

Irradiation has always been a useful means of palliating patients with advanced cancer having symptomatic localized disease.

With potentially curative radiotherapy now being done more frequently in larger centers, the community hospitals treat a large percentage of their therapy patients palliatively. This is a valuable service, particularly if attention is paid to the possibilities for short courses of treatment.

Practical new approaches to palliative therapy will be discussed and rational treatment schemes for certain treatment situations will be outlined.

COURSE 301

Thursday

LUCY FRANK SQUIRE, M.D.
New York, New York

SPENCER BORDEN, IV, M.D.
Boston, Massachusetts

DAVID KUMPE, M.D.
Boston, Massachusetts

WILLIAM M. COLAIACE, M.D.
Medford, Massachusetts

Teaching Methods in Undergraduate Radiology

Various methods used in teaching radiology to medical students will be discussed and demonstrated. A pilot group of students will be on the stage and the audience itself will participate in the demonstration.

A new self-instructional method being used presently will be demonstrated also, and data will be offered on a testing procedure for evaluating teaching methods.

COURSE 303
and
COURSE 403

Thursday and Friday

NORMAN E. CHASE, M.D.
New York, New York

IRWIN I. KRICHEFF, M.D.
New York, New York

Course 303. Angiographic Investigation of Supratentorial Tumors

The first session of this two part course on neuroradiology will be devoted to the angiographic investigation of supratentorial tumors. Following a brief review of the technical aspects and anatomy, a detailed logical approach to the diagnosis and localization of supratentorial tumor masses will be presented. Specific changes that should lead one to the diagnosis and accurate placement of tumors will be reviewed. The course will include an analysis of arterial and venous displacements as well as dynamic changes to assist the student not only in the diagnosis and localization of masses, but also in the angiographic changes which frequently lead to a specific pathologic diagnosis.

Course 403. Vascular Diseases of the Central Nervous System

The second session will be devoted to vascular diseases of the central nervous system. The angiographic evaluation of intracranial aneurysms and arteriovenous malformations will be covered in detail. Technical aspects, diagnosis and the determination of the site of bleeding in the presence of multiple lesions, as well as the rationale and value of postoperative angiography will be included. Cerebral vascular occlusive disease, both extracranial and intracranial, will comprise the remainder of the presentation. Techniques of investigation, analysis of stenoses, occlusions and collateral pathways as well as physiologic changes will be presented. The practical clinical value of angiography, and the factors for and against performing angiography will be discussed.

COURSE 304
and
COURSE 404

Thursday and Friday

RICHARD H. MARSHAK, M.D.
New York, New York

Course 304. Small Bowel

1. Malabsorption, including immunoglobulin disorders
2. A group of disorders about which our knowledge is increasing, including retractile mesenteritis, eosinophilic gastroenteritis, Henoch's purpura, amyloidosis and light and heavy chain disorders
3. Tumors of the small bowel
4. Inflammatory lesions of the small bowel

Course 404. Colon

1. Technique
2. Granulomatous colitis and its differential diagnosis from diverticulitis
3. Multiple polypoid lesions of colon. These include a variety of lesions which must be differentiated from one another
4. Hemorrhagic disorders
5. Ischemia of the colon
6. Stasis and development of ulceration and carcinoma

COURSE 305
and
COURSE 405

Thursday and Friday

MAURICE D. SACHS, M.D.
Cleveland, Ohio

Arthrography of the Knee Joint

Arthrography is a valuable diagnostic aid in studying internal derangements of the knee joint. The author's experience with this procedure stretches over 25 years, is a continuing process, and includes more than 10,000 examinations.

In this 2 day course the anatomy of the knee joint, the advantages and disadvantages of contrast media, and the roentgen technique will be illustrated and discussed.

Normal and abnormal arthrograms will also be illustrated and discussed, in detail.

In the abnormal group, in addition to meniscal tears, examples of "cyst-discoid" meniscus, synovitis, chondromalacia, and fat pad changes will be presented.

COURSE 309
and
COURSE 409

Thursday and Friday

ELIAS G. THEROS, M.D.
Washington, D. C.

Course 309. Pulmonary Pseudotumors and Mesotheliomas

When the radiologist is confronted with a large mass in the lung fields, he usually considers a solitary metastasis, a primary bronchogenic carcinoma, a lung sarcoma, or perhaps an unusual granuloma. This course will deal with 2 other lesions which often present this way.

The inflammatory pseudotumor of the lung is a lesion that is little known to roentgenologists, since it has only occasionally been described in the roentgenologic literature. Yet it is of sufficient frequency so that it is important in differential diagnosis. The entity will be defined and its roentgenographic and pathologic features illustrated, drawing from our AFIP experience of over 100 cases.

Mesotheliomas are better known, particularly because of their association with the increasing amounts of asbestos in our environment.

Their increasing importance justifies a closer look at their roentgenologic and pathologic characteristics and especially their features in differential diagnosis.

Course 409. Lesions of the Mediastinum

The radiologist has long been aware that the best strategy in the evaluation of mediastinal lesions is to rely on the statistics of location.

He has learned, for example, that thymic and teratoid tumors are almost invariably found in the anterior mediastinum and that neurogenic tumors find their home in the posterior mediastinum. In addition, he has observed that most masses in the middle mediastinum are due to lymphadenopathy resulting from inflammation or neoplasia.

It is also true, however, that he can differentiate many of the lesions that tend to occur in a particular mediastinal compartment by increasing his awareness of their roentgen morphologic characteristics, such as their size, shape, contour, density, and the like.

This awareness can be most effectively developed by the study and correlation of such roentgen findings with gross anatomic specimens, and this will be a feature of this presentation.

COURSE 311

Thursday

MILTON ELKIN, M.D.
Bronx, New York

Radiologic Considerations of Trauma to the Urinary Tract

It is the radiologist's responsibility to define the extent of injury to the kidney as an important aid in the proper management of the patient. Intravenous urography, the primary study up to now, has often been inadequate. In many instances angiography is the critical radiologic study. Criteria will be defined for the use of angiographic study in suspected renal trauma, exemplified by illustrative cases.

Ureteral, bladder and urethral injuries will also be discussed with consideration of mechanisms of injury, diagnostic criteria and clinical significance.

COURSE 312

Thursday

W. B. YOUNG, M.D.
London, England

Primary Malignant Tumors of Bone

The classification of primary malignant tumors of bone will be discussed.

An attempt will then be made to demonstrate the more characteristic radiologic features of individual types of tumor, both common and uncommon, and less typical forms will be shown.

Problems in diagnosis will be discussed with special regard to differentiation of certain aggressive forms of benign tumors, metastases and infections of bone.

COURSE 313

Thursday

HAROLD SNOW, D.V.M.
Los Angeles, California

Diagnostic Procedures for a Radiology Research Laboratory Environment

The success of biomedical experimentation often begins with the well contemplated choice of the experimental animal model. For instance, the pig is a popular animal model because of the anatomic similarity of its viscera to that of man. Selection of subhuman primates is desirable, but extremely expensive, and the use of dogs and cats in biomedical research is often one of convenience due to the relative ease of procurement and low cost.

Selection of the anesthetic in specific experimental situations, pharmacodynamics and modes of administration on several animal species will be covered in depth as well as accepted methods of handling and restraint of experimental animals. Principles of preoperative, postoperative, and postangiographic care will be described in context with clinical laboratory findings.

Animal research radiologic techniques are demonstrated including the recording of electrocardiograms, pressure and flow tracings, and accessory laboratory equipment such as the Bennett respiratory emergency resuscitation equipment and pressure contrast material injector.

COURSE 314

Thursday

S. BOYD EATON, JR., M.D.
Boston, Massachusetts

JOSEPH T. FERRUCCI, M.D.
Boston, Massachusetts

Radiology of the Pancreas and Duodenum

The pancreas is no longer a "hidden organ." Once symptoms develop, a radiologic diagnosis can be made in the great majority of instances. The variety of diagnostic techniques available is not an indictment of their effectiveness, but rather an indication that the radiologic work-up must be carefully tailored to the clinical manifestations at hand.

This course analyzes the information available from plain roentgenograms, barium studies, cholangiography, isotopic imaging and angiography. Since examination of the duodenum is of value in nearly all patients with suspected pancreatic disease, intrinsic duodenal pathology as well as abnormalities produced by pancreatic disorders will be presented and differential diagnostic considerations stressed.

The over-all goal of the course is development of a comprehensive strategy for radiologic investigation of pancreatico-duodenal disease.

COURSE 315

Thursday

ROBERT A. NEBESAR, M.D.
Boston, Massachusetts

JAMES J. POLLARD, M.D.
Boston, Massachusetts

Angiography of the Abdomen

This course offers a comprehensive discussion of visceral angiography with particular reference to the liver, pancreas, spleen and bowel in adults and children.

The proper techniques for optimal arterial visualization of these organs and for demonstration of the portal venous system will be briefly discussed.

Anatomic variations and their relevance to the proper performance and correct interpretation of angiograms and preoperative assessment of various conditions will be covered.

The major portion of the presentation will include a systematic demonstration of angiography in the various pathologic conditions affecting the liver, spleen, pancreas and bowel.

An attempt will be made to place in perspective the indications for and relative value of angiography in diagnosis.

Among the conditions which will be discussed are: hepatic neoplasm (benign and malignant); hepatic inflammation (including cirrhosis); hepatic and splenic trauma; pancreatic neoplasms and inflammations; gastrointestinal bleeding; and conditions affecting the portal venous system.

COURSE 316

Thursday

ROBERT H. SAGERMAN, M.D.
Syracuse, New York

Radiation Therapy for Childhood Malignancy

The role of radiation therapy, surgery and chemotherapy in the management of childhood malignancies will be reviewed critically in terms of long-term survival (cure), local tumor control, and palliation.

Long-term effects of each treatment mode will be assessed.

A rational approach to each primary neoplastic site will be presented and areas requiring further study indicated.

Time will be reserved for questions and technical details.

COURSE 401

Friday

LEWIS E. ETTER, M.D.
Pittsburgh, Pennsylvania

Examination of Plain Film Roentgenograms of the Skull with some Illustrations of Pathologic Alterations

This will be an examination in depth identifying many previously confusing lines and shadows based upon studies of disarticulated skull components and the Spalteholz Transparent Skull. Each bone will be studied in several projections with analysis of its basic roentgen anatomy. Emphasis will be placed on variations seen in slightly differing positions of individual components in relation to the whole skull.

To determine the details of the roentgen anatomy of the skull, various component bones of dry skulls were disarticulated and roentgenograms were made of each, first posed separately in various standard projections, then placed in the respective skulls similarly oriented for routine roentgenography. In this way it has been possible to determine exactly which feature each of the cranial bones studied contributes to the image of the intact skull.

Both cranial and facial components will be examined with attention to the radiologist's daily needs. After demonstrating the normal roentgen anatomic features as above outlined, a few pathologic alterations will be shown.

COURSE 402

Friday

JACK EDEIKEN, M.D.
Philadelphia, Pennsylvania

Metabolic Bone Disease

The *sine qua non* of metabolic bone disease is density change.

Bone densitometry will be discussed during the first portion of this course. Thereafter, the x-ray change of osteoporosis and osteomalacia will be considered.

Diseases which epitomize these changes will be used to illustrate the important points in diagnosis.

Other diseases which may not be metabolic will be included in the differential diagnosis and discussion.

COURSE 406

Friday

SIDNEY WALLACE, M.D.
Houston, Texas

Lymphangiography Today

Lymphangiography has an established position in the diagnosis and management of patients with lymphoma. The reliable demonstration of metastatic disease from carcinoma of the uterine cervix, ovary, testicle, colon, etc. has permitted more accurate staging and, therefore, modified the therapeutic approach. There is excellent correlation with positive findings.

Cirrhosis, constrictive pericarditis, edema, protein losing enteropathy and chylous reflux are associated with altered lymphatic dynamics. Any change in blood flow is accompanied by a concomitant change in lymphatic dynamics.

Alterations in lymphatic dynamics, modes of metastases, and lymphangiographic interpretation will be reviewed.

COURSE 407

Friday

BERNARD S. WOLF, M.D.
New York, New York

The Distal Esophagus

Controversy over criteria for the diagnosis of a small sliding hiatal hernia cannot be resolved until there is agreement on the definition of such a hernia.

The currently quoted definition of a sliding hernia—"stomach above the hiatus"—is unsatisfactory because there is no single generally accepted landmark for the esophagogastric junction. Another definition is suggested—"a hiatal hernia is present if the attachments of the esophagogastric region to the diaphragm are loose or long and there is compromise of the normal anti-reflux mechanism."

The viewpoint to be presented is of importance because of recent clinical emphasis on "reflux without a hernia" or "primary incompetence of the esophagogastric sphincter." To demonstrate this condition, the radiologist is asked to concentrate on maneuvers to induce reflux. Reflux may, however, be demonstrable in individuals with few or no pertinent symptoms if vigorous efforts are made to produce it or if excessive emphasis is placed on a positive water-swallowing or "siphonage" test. Significant surgically correctable reflux should not be assumed to be present unless there is roentgen evidence of a sliding hernia according to the definition given above.

Exceptions to this statement may occur but rarely without special circumstances such as early scleroderma or Raynaud's disease, after excessively forceful dilatation for achalasia, after myotomy for achalasia or a "hypertensive sphincter," or as a result of drugs such as vincristine which paralyzes the sphincteric mechanism.

In the absence of such factors, the significance of reflux demonstrated by roentgen methods must be confirmed by other evidence such as pH measurements in the esophagus after instillation of acid in the stomach and measurements of resting sphincter pressure and sphincter responses.

Operative intervention to control reflux on the basis of equivocal symptoms fortified by reflux induced by provocative maneuvers during a barium meal examination is not justified. On the other hand, heartburn due to minimal herniation can be treated well by currently available operative procedures. If symptoms are intractable, operative intervention is indicated and should be curative.

An effort will be made to describe the roentgen features indicative of minimal herniation as well as the features of asymptomatic hernias. The roentgen criteria of a satisfactory hiatal hernia repair will also be discussed.

COURSE 408

Friday

ERNEST J. FERRIS, M.D.
Boston, Massachusetts

PETER R. CLAPP, M.D.
Boston, Massachusetts

Retroperitoneal Diagnosis

This course will focus on some disease processes of retroperitoneal organs including the pancreas, adrenal glands, kidney, retroperitoneal connective tissue, lymph nodes, the aorta and the inferior vena cava. The value of plain film roentgenography, intravenous pyelography, barium studies, as well as special examinations such as percutaneous transhepatic cholangiography, duodenography, ultrasound, arteriography, venography, and lymphography will be discussed.

The salient points of the most important radiologic examinations for each organ will be discussed as below.

Pancreas

plain roentgenogram
barium studies
duodenography
cholangiography
arteriography
venography

Adrenals

arteriography
venography

Kidney

plain roentgenogram
intravenous pyelogram
arteriography
venography
ultrasound

Soft tissues

plain roentgenogram
intravenous pyelogram
arteriography of small vessels

Lymph nodes

lymphography
venography
arteriography

Vascular structures

primary and secondary diseases
vena cava
aorta

COURSE 410

Friday

MARVIN M. D. WILLIAMS, Ph.D.
Rochester, Minnesota

Preparing for an Examination in Radiological Physics

The answers given to various types of questions in radiological physics, although not entirely incorrect, are often unsatisfactory. Frequently, the answer given is an equation, a numerical constant, or a memorized statement.

The examinee frequently cannot tell: (1) what conditions must be satisfied in order that the use of the equation is justified; (2) what the numerical constant represents; or (3) what the memorized statement means.

This problem and some of these questions will be discussed with the aim not only of indicating a correct answer, but also of showing that an acceptable answer is likely to follow naturally when the subject matter is understood.

The discussion will include suggestions from other examiners in radiological physics.

COURSE 411

Friday

BERNARD S. ARON, M.D.
Cincinnati, Ohio

Radiation Biology for the Clinical Radiation Therapist

Clinical radiation therapy has developed empirical histologic concepts of radiation sensitivity, tumor cell growth, dose-time effects, etc. Advances in radiation biology serve as a basis for understanding these clinical observations. These advances will be examined as they are pertinent to the radiation therapist.

The "inherent" radiation sensitivity of cells will be discussed in terms of cell cycle, effect on D.N.A., cell survival curves, and radiation sensitization and protection.

Many factors modify this inherent radiation sensitivity. The factors of "apparent" sensitivity which will be outlined include: dose-time effects (fractionation and protection); oxygen effect (hyperbaric oxygen, air oxygen breathing, anoxia, and fractionation); concentration of energy transfer events (use of neutrons and pi mesons); and cell population kinetics (normal and tumor cell growth, and response to radiation).

The radiation biologic basis for single and fractionated radiation treatments will be discussed.

SECTION ON INSTRUCTION

ORDER SHEET

It is important to register for the Instruction Courses as early as possible since the number admitted to each course will be limited. *It is also very important that first, second and third choices be listed for each period.* All orders for tickets will be filled according to the postmark on the envelopes.

Nonmembers of the American Roentgen Ray Society will pay $3.00 for each course-period or a maximum of $10.00 for 4 courses. *Nonmembers' fees must accompany this Order Sheet and will not be returned unless cancellation is received before September 17, 1971.*

Members of the American Roentgen Ray Society, residents in Radiology, and nonmembers who are contributing to the program, either by way of an instruction course, a paper or a *scientific* exhibit, are not required to pay for these courses but must fill out these Pink Order Sheets, indicating their choices.

Tickets for courses will be mailed to each registrant whose order reaches the office of the Director on or before September 17, 1971. They will be sent to the address below unless registrant directs otherwise.

Please fill out the following (type or print):

....
Last Name First Name or Initials

....
Street Address

....
City State Zip Code

Check
☐ Member
☐ Guest
☐ Resident in Radiology at: ....
☐ Scientific Exhibitor/Paper

Those whose advance registration reaches the office of the Director after September 17, 1971 may obtain their tickets at the Registration Desk of the Section on Instruction on Monday afternoon, September 27, 1971 and thereafter. At this time tickets may also be obtained for courses not sold out.

For convenience in selecting your courses consult the condensed schedule of courses.

BE SURE TO FILL OUT SECOND AND THIRD CHOICES FOR EACH PERIOD

Period	First Choice		Second Choice		Third Choice	
	Course No.	Instructor	Course No.	Instructor	Course No.	Instructor
Tuesday						
Wednesday						
Thursday						
Friday						

Send this order to:

DR. HAROLD O. PETERSON, 1995 West County Road "B", St. Paul, Minnesota 55113
OR if you are mailing late, send to:
DR. HAROLD O. PETERSON, The Sheraton-Boston Hotel,
Boston, Massachusetts 02199

CONDENSED SCHEDULE OF COURSES

TUESDAY **3:00 to 4:30 P.M.** **September 28, 1971**

101—**Jacobson**
Benign Bone Tumors: Interesting Manifestations

102—**Macmillan**
Sinuses and Orbits

103—**Nelson**
The Discovery of Gastric Ulcers and the Differential Diagnosis Between Benignancy and Malignancy

104—**Grigg**
The History of Radiology I

105—**McConnell**
Lane
Nuclear Medicine

106—**Heitzman**
Roentgen Pathologic Correlations in Common Pulmonary Disorders

107—**McAfee**
Current Status of Radioisotopic Methods in the Diagnosis of Renal Disease

108—**Lalli**
The Tailored Urogram

109—**Meyers**
Intestinal Obstruction and the Detection of Small Amounts of Intraperitoneal Fluid

110—**Shopfner**
Current Status of Pediatric Uroradiology

111—**Singleton**
Pediatric Pulmonary Abnormalities

112—**Barnhard**
The Use of Computers in Diagnostic Radiology

113—**New**
Bauman
Pendergrass
Hydrocephalus—Present Status of Radiological and Radionuclide Investigation

114—**Meszaros**
The Pulmonary Circulation and Pulmonary Vessels

115—**Wise**
Current Concepts of Cholangiography

116—**Kligerman**
Tricks and Treatment Planning

CONDENSED SCHEDULE OF COURSES

WEDNESDAY **3:00 to 4:30 P.M.** **September 29, 1971**

201—**Jacobson**
Some Interesting Features of Skeletal Radiology: Challenges in Diagnosis

202—**Macmillan**
Temporal Bone

203—**Nelson**
Fundamentals in the Roentgenologic Study of Skeletal Trauma

204—**Grigg**
The History of Radiology II

205—**McConnell**
Lane
Nuclear Medicine

206—**Gwinn**
Roentgen Manifestations of Acquired Bone Disease in Children

207—**Potts**
Pneumoencephalography

208—**Dunbar**
Pediatric Chest Radiology

209—**Judkins**
Selective Coronary Arteriography

210—**Felson**
Diffuse Lung Lesions

211—**Potter**
Sialography

212—**Seaman**
Radiology of the Larynx and Pharynx

213—**Tuddenham**
The Perception of the Roentgen Shadow: Its Mechanisms; Its Uncertainties; Its Control

214—**J. Wolfe**
Mammography

215—**Margulis**
Radiology of Colonic Polyps

216—**Caldwell**
Practical Palliative Radiotherapy

CONDENSED SCHEDULE OF COURSES

THURSDAY **3:00 to 4:30 P.M.** **September 30, 1971**

301—**Squire**
Borden
Kumpe
Colaiace
Teaching Methods in Undergraduate Radiology

302—**Macmillan**
Larynx, Pharynx, Cervical Esophagus and Trachea

303—**Chase**
Kricheff
Angiographic Investigation of Supratentorial Tumors

304—**Marshak**
Small Bowel

305—**Sachs**
Arthrography of the Knee Joint

306—**Heitzman**
The Mediastinum: Roentgen Anatomic Correlations

307—**Potts**
Pneumoencephalography

308—**Dunbar**
Pediatric Radiology of the Gastrointestinal Tract

309—**Theros**
Pulmonary Pseudotumors and Mesotheliomas

310—**Felson**
Abdominal Gasmanship

311—**Elkin**
Radiologic Considerations of Trauma to the Urinary Tract

312—**Young**
Primary Malignant Tumors of Bone

313—**Snow**
Diagnostic Procedures for a Radiology Research Laboratory Environment

314—**Eaton**
Ferrucci
Radiology of the Pancreas and Duodenum

315—**Nebesar**
Pollard
Angiography of the Abdomen

316—**Sagerman**
Radiation Therapy for Childhood Malignancy

CONDENSED SCHEDULE OF COURSES

FRIDAY **1:30 to 3:00 P.M.** **October 1, 1971**

401—**Etter**
Examination of Plain Film Roentgenograms of the Skull with some Illustrations of Pathologic Alterations

402—**Edeiken**
Metabolic Bone Disease

403—**Chase**
Kricheff
Vascular Disease of the Central Nervous System

404—**Marshak**
Colon

405—**Sachs**
Arthrography of the Knee Joint

406—**Wallace**
Lymphangiography Today

407—**B. Wolf**
The Distal Esophagus

408—**Ferris**
Clapp
Retroperitoneal Diagnosis

409—**Theros**
Lesions of the Mediastinum

410—**Williams**
Preparing for an Examination in Radiological Physics

411—**Aron**
Radiation Biology for the Clinical Radiation Therapist

JAMES PICKER FOUNDATION—AWARDS PROGRAM FOR 1971-1972

NATIONAL RESEARCH COUNCIL, NATIONAL ACADEMY OF SCIENCES

On behalf of the James Picker Foundation, the National Research Council on June 3, 1971, announced the awarding of 35 fellowships and grants to medical scientists for support of research and training in radiology and nuclear medicine.

The Awards Program, valued this year at approximately $493,284, is supported by the James Picker Foundation and administered by the Committee on Radiology of the Research Council's Division of Medical Sciences. Twenty-eight of the awards are renewals for work already in progress, while 7 are being given for the first time.

Those selected to participate in the program for the 1971–1972 academic year are:

Advanced Fellowships in Academic Radiology. Dr. Ronald G. Evens, St. Louis, Missouri; Dr. Reginald E. Greene, Boston, Massachusetts; Dr. A. Everette James, Baltimore, Maryland; Dr. Arvin E. Robinson, Durham, North Carolina; and Dr. Milos Sovak, Philadelphia, Pennsylvania.

Research Fellowships. Dr. Vicente J. Caride, New Haven, Connecticut; Dr. Gordon Gamsu, San Francisco, California; Dr. James Hevezi, Madison, Wisconsin; Dr. W. Barrie Jones, Oslo, Norway and Middlesex, England; Dr. Max S. Lin, Berkeley, California; Dr. Patrick J. McManamon, Newfoundland, Canada; Dr. Robert J. Nickles, Madison, Wisconsin; and Dr. Martin H. Schneiderman, Gainesville, Florida.

Awards for Scholars in Radiological Research. Dr. Robert J. Cowan, Winston-Salem, North Carolina; Dr. George C. Curry, Dallas, Texas; Dr. Michael A. Davis, Boston, Massachusetts; Dr. Eli Glatstein, Stanford, California; Dr. Warren A. Hinchcliffe, San Francisco, California; Dr. Paul B. Hoffer, Chicago, Illinois; Dr. Robert W. Parkey, Dallas, Texas; Dr. Lee B. Talner, San Diego, California; and Dr. Robert I. White, Jr., Baltimore, Maryland.

Research Grants. Dr. Edward G. Bell, Syracuse, New York; Dr. Geoffrey T. Benness, Sydney, Australia; Dr. James D. Collins, Los Angeles, California; Dr. Allan E. Dumont, New York, New York; Dr. Raymond Gramiak, Rochester, New York; Dr. William N. Hanafee, Los Angeles, California; Drs. James G. Kereiakes and Alvin Shapiro, Cincinnati, Ohio; Dr. James H. Larose, Atlanta, Georgia; Dr. Ralph M. Lilienfeld, New York, New York; Dr. William H. Northway, Jr., Stanford, California; Dr. Howard J. Ricketts, Seattle, Washington; Dr. Margot R. Roach, Ontario, Canada; Dr. Shih H. Tsai, Minneapolis, Minnesota; and Dr. William J. Tuddenham, Philadelphia, Pennsylvania.

Applications for 1972–1973 should be submitted by *October 1, 1971.* Application blanks and further details may be obtained from the *Committee on Radiology, National Academy of Sciences-National Research Council, 2101 Constitution Avenue, N.W., Washington, D.C. 20418.*

The requirements for application are identical to those published in the July, 1970 issue of this JOURNAL (p. 646).

NEWS ITEMS

OFFICERS OF THE COUNCIL OF SECTION ON RADIOLOGY OF THE AMERICAN MEDICAL ASSOCIATION

The American Medical Association in its desire to bring about a closer collaboration with the various Specialty Societies has created the *Councils of Sections* consisting of Representatives of such Societies, based on numerical membership. These Councils are replacing the former Executive Committees of each Section, of which presently there are 23. The Council of the Section on Radiology will have 7 members: 3 representing the American College of Radiology; 2 the Radiological Society of North America; and 1 each the American Society of Nuclear Medicine and the American Roentgen Ray Society.

The Representative of the American Roentgen Ray Society is Dr. Richard G. Lester of Durham, North Carolina.

The Officers of the Council are: *Chairman*, Dr. John R. Hodgson, Rochester, Minnesota; *Vice-Chairman*, Dr. E. James Potchen, St. Louis, Missouri; *Secretary*, Dr. Antolin Raventos, Davis, California; *Exhibit Representative*, Dr. Richard G. Lester, Durham, North Carolina; *Section Delegate to the House of Delegates*, Wendell G. Scott, St. Louis, Missouri; and *Section Alternate Delegate to the House of Delegates*, Seymour F. Ochsner, New Orleans, Louisiana. The seventh member of the Council is Dr. Richard H. Chamberlain, Philadelphia, Pennsylvania, the Chairman of the former Executive Committee, whose term expires December 31, 1971.

The Officers of the new Council will begin their terms January 1, 1972.

Dr. Ted Leigh, Atlanta, Georgia, is the program representative and Dr. Glenn E. Sheline, San Francisco, California, the assistant program representative. Drs. Leigh and Sheline are not members of the Council.

The next Annual Meeting of the American Medical Association will be held in San Francisco, California, June 18–22, 1972.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS ELECTS NEW OFFICERS

At the Annual Meeting of the Association of University Radiologists, held in Durham, North Carolina on May 13, 1971, the following slate of officers was elected: *President*, James H. Scatliff, M.D., Department of Radiology, University of North Carolina, School of Medicine, Chapel Hill, North Carolina 27514; *President-Elect*, Elliott C. Lasser, M.D., Department of Radiology, University Hospital of San Diego County, San Diego, California 92103; and *Secretary-Treasurer*, Byron G. Brogdon, M.D., Department of Radiology, University of New Mexico, School of Medicine, Albuquerque, New Mexico 87106.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS (AUR)

Annual Memorial Medal Award

The Association of University Radiologists wishes again to announce that an Annual Memorial Medal Award will be given to the radiology resident writing the best original paper. The paper can represent the work of one or more investigators, but the resident submitting the paper must qualify as the senior author.

Manuscripts to be considered for the 1972 competition must be received by March 1, 1972. Inquiries should be addressed and papers submitted to: Byron G. Brogdon, M.D., Department of Radiology, University of New Mexico, School of Medicine, Albuquerque, New Mexico 87106.

The winner of the competition will be invited to present his work at the Annual Meeting of the AUR and will receive a monetary award in addition to the medal. The next AUR Annual Meeting will be in

Albuquerque, New Mexico, May 11–13, 1972.

In the 1971 competition, co-winners were selected. These were: Dr. C. Richard Dunn of the Radiology Department of the University of California, San Diego, School of Medicine, for a paper titled, "The Pharmaco-Kinetics of Telepaque Metabolism: The Relation of Blood Concentration and Bile Flow to the Rate of Hepatic Excretion," and Dr. Melvyn Korobkin of the Radiology Department of the University of California, San Francisco, School of Medicine, for a paper titled, "The Nephrogram of Hemorrhagic Hypotension."

SYMPOSIUM ON DIAGNOSTIC RADIOLOGY OF URINARY TRACT

The Department of Diagnostic Radiology of the Lahey Clinic Foundation will sponsor a "Symposium on the Diagnostic Radiology of the Urinary Tract," to be held at the Sheraton-Plaza Hotel, Boston, Massachusetts, September 17 and 18, 1971.

The Faculty will include: Stanley Baum, M.D., Franca S-R Danforth, M.D., Joseph B. Dowd, M.D., Ernest J. Ferris, M.D., Lloyd D. Flint, M.D., Alexander Gottschalk, M.D., W. Thorne Griscom, M.D., Anthony F. Lalli, M.D., John A. Libertino, M.D., Thomas F. Meaney, M.D., Harry Z. Mellins, M.D., Charles E. Shopfner, M.D., Robert E. Wise, M.D., and Leonard Zinman, M.D.

For further information please contact: Robert E. Wise, M.D., Chairman, Department of Diagnostic Radiology, Lahey Clinic Foundation, 605 Commonwealth Avenue, Boston, Massachusetts 02215.

SYMPOSIUM ON POLYTOMOGRAPHY OF THE TEMPORAL BONE

The Fourth Symposium on Polytomography of the Temporal Bone will be given under the auspices of The Wright Institute of Otology at Community Hospital, Indianapolis, Indiana, on October 9 and 10, 1971.

The subjects covered are: Basic anatomy of the temporal bone; and technique of polytomography of the temporal bone, with demonstrations of normal tomograms.

Pathologic conditions revealed by polytomography, such as cholesteatoma, ossicular chain problems, otosclerosis, fractures, foreign bodies, tumors, and congenital anomalies will be shown on original tomograms and the clinical applications discussed.

The number of registrants is limited to 18.

Inquiries should be directed to: The Wright Institute of Otology, Inc., Community Hospital of Indianapolis, Inc., 1500 North Ritter Avenue, Indianapolis, Indiana 46219.

CLINICAL NUCLEAR MEDICINE COURSE

This course, which will be held October 28–30, 1971, is designed for the physician practicing nuclear medicine in the community. The interpretation of older and better known procedures will be reviewed; a number of newer procedures, still not common outside the major medical centers but which are feasible in community hospitals, will be discussed.

A special feature of the program will be several sessions devoted to practical nuclear medicine instrumentation: the pros and cons of scanners and cameras will be discussed, as well as what to look for when buying equipment. A thorough market review of available equipment is planned.

The 2½ day course will consist of daily sessions with heavy clinical orientation; 2 evening workshops will center around instrumentation.

A Guest Faculty, as well as the University of Michigan Medical Center staff, will participate.

Further information may be obtained from Dr. Harry A. Towsley, Chairman, Department of Postgraduate Medicine, Towsley Center, University of Michigan Medical Center, Ann Arbor, Michigan 48104.

THIRTEENTH SYMPOSIUM ON NUCLEAR MEDICINE

Oak Ridge Associated Universities (ORAU) Medical Division

The Medical Division of ORAU will present its Thirteenth Symposium on Nuclear

Medicine, November 15–19, 1971, in Oak Ridge, Tennessee, on the topic, "Clinical Uses of Radionuclides: Critical Comparison with Other Techniques."

The Oak Ridge program is being offered for the first time this year in cooperation with the American College of Physicians as one in the College's annual series of post-graduate courses in various specialty fields. Papers and discussions will be directed toward the practicing physician and are designed to help him evaluate the value of diagnostic methods using radioisotopes as compared with radiologic techniques and other laboratory tests.

Presentations by 35 invited speakers will cover applications of these techniques in neurology, gastroenteroloy, hematology, endocrinology, rheumatology, cardiology, and nephrology. The diagnosis of cancer will be emphasized.

Further information may be obtained from: Edward C. Rosenow, M.D., Executive Director, American College of Physicians, 4200 Pine Street, Philadelphia, Pennsylvania 19104.

A COMPREHENSIVE COURSE IN NEURORADIOLOGY

New York University Post-Graduate Medical School

This course will be held November 1–5, 1971, under the Directorship of Irvin I. Kricheff, M.D., Professor of Radiology, New York University Post-graduate Medical School.

The curriculum will include: regular and tomographic examination of the skull and spine; cerebral angiography; pneumoencephalography and ventriculography; studies of the spinal cord by myelography and angiography; brain scanning and other isotope studies; and technical aspects and special techniques.

The Guest Faculty includes: Dr. René Djindjian, Paris, France; Dr. Ralph Heinz, Pittsburgh, Pennsylvania; Dr. Sadek K. Hilal, New York, New York; Dr. Yun Peng Huang, New York, New York; Dr. Stephen A. Kieffer, Minneapolis, Minnesota; Dr. Norman E. Leeds, New York, New York; Dr. Thomas H. Newton, San Francisco, California; Dr. Gordon D. Potts, New York, New York; Dr. Mannie M. Schechter, New York, New York; Dr. Juan M. Taveras, St. Louis, Missouri; and Dr. Ernest H. Wood, New York, New York.

A detailed brochure of the course will be available in September. Owing to space limitations, early registration is advised.

For application please write: Office of the Recorder—Room 158 MSB, New York University, Post-Graduate Medical School, 550 First Avenue, New York, New York 10016.

SYMPOSIUM ON MALIGNANT TUMORS IN CHILDHOOD

The Fifth Annual Radiotherapy Symposium on "Recent Advances in Diagnosis and Therapy of Malignant Tumors in Childhood," sponsored by the University of Miami School of Medicine and the Department of Radiation Therapy, Cedars of Lebanon Hospital, will be held in Miami at the Sheraton-Four Ambassadors Hotel on December 10 and 11, 1971.

For further information, please write to Mario M. Vuksanovic, M.D., Director, Department of Radiation Therapy, Cedars of Lebanon Hospital, Miami, Florida 33125.

SOCIÉTÉ FRANÇAISE DE NEURORADIOLOGIE

The Société Française de Neuroradiologie will hold its Annual Meeting in December 1971 in Paris.

The chosen theme is "Neuroradiologic Study of the Cavernous Sinus," which will be considered in 3 parts: (1) anatomy and radiologic anatomy; (2) technique of exploration of the cavernous sinus; and (3) pathologic lesions of the cavernous sinus (vascular, tumoral, and infectious).

For further information please write to the Secretary General of S.F.N.R., Pr. Ag. R. Djindjian, 16 rue de l'Université, Paris 7o, France.

COURSE IN ADVANCED NEURORADIOLOGY

The Normal and Abnormal Phlebogram in Vertebral Angiography

This Course will be held under the aegis of Professor F. Isch, Dean of the Faculty of Medicine, in Strasbourg, France, March 10 and 11, 1972.

The participants will be divided into small groups. Each group will attend 18 tutorials.

Applications for enrollment should be sent to the Course Secretary: Professor A. Wackenheim—Service de Neuroradiologie du CHU, 1, place de l'Hôpital—67 Strasbourg, France.

INTERNATIONAL SYMPOSIUM ON CEREBRAL BLOOD FLOW REGULATION, ACID-BASE AND ENERGY METABOLISM IN ACUTE BRAIN INJURIES

This International Symposium will be held in Rome and Siena, October 28–31, 1971.

The Organizing Committee is formed by: C. Fazio, C. Fieschi, A. Agnoli, N. Battistini, L. Bozzao and A. Carpi.

For further details please write to: Prof. Cesare Fieschi, Clinica delle Malattie Nervose e Mentali dell'Università di Siena, Siena, Italy.

It is with deep regret that we announce the death of Hermann Holthusen, Prof. Dr. med., Dr. med. h.c., and Dr. rer. nat. h.c., on May 7, 1971, after a long illness in Hamburg, West Germany, at the age of 85 years.

Dr. Holthusen has been an Honorary Member of the American Roentgen Ray Society since 1939.

BOOK REVIEW

Urology. Third edition. Three Volumes. Edited by the late Meredith F. Campbell, M.S., M.D., F.A.C.S., Late Professor of Urology, New York University and Consulting Urologist, Bellevue Hospital, New York, N. Y.; and J. Hartwell Harrison, M.D., F.A.C.S., Cutler Professor of Surgery (Urology), Harvard Medical School, and Chief of Urology, Peter Bent Brigham Hospital, Boston, Mass., with 74 contributors. Cloth. Pp. 2,888, with 1,769 illustrations. Price per set $110.00 U.S., $118.80 Canada. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1970.

This is the third edition of the three-volume text, *Urology*, first published in 1954. Dr. Campbell brought out the first two editions alone but became ill in 1968 and was joined by Dr. Harrison as co-editor before this third edition was completed.

The organization and balance of the earlier editions are preserved. The clinical practice of urology is presented within the framework of basic science. Revisions and 7 new chapters have been added without increasing the number of pages. Ten new authors raise the number of contributors to 74. Accordingly, topics overlap and duplication is at times tedious, although there is individual variation providing some compensation. Seventy chapters, including 6 in the pre-clinical section of anatomy and physiology, contain with an encyclopedic scope the common and unusual diseases, which should allow *Urology* to easily maintain its eminence as the best comprehensive English reference textbook.

Although my over-all response is one of admiration, a few comments are offered in hope of influencing the next edition. The radiologic discussion in the section of examination and diagnosis is disappointing. It should either be omitted or be adequately and accurately portrayed in the future. For example, of 25 pages partially devoted to uroradiology, renal arteriography by the translumbar aortographic method receives 5 pages and the selective femoral catheter technique is covered in one paragraph. No references in this section are more recent than 1958 and this cannot be dismissed as being a victim of publication lag.

This edition is too large and expensive for the student of Radiology. However, it is strongly recommended as a comprehensive reference work and should be a valuable addition to the departmental library.

Richard C. Pfister, M.D.

BOOKS RECEIVED

Cerebellum of the Rhesus Monkey: Atlas of Lobules, Laminae, and Folia, in Sections. By John C. Madigan, Jr., M.D., Intern in Surgery, Presbyterian Hospital, New York, N. Y.; and Malcolm B. Carpenter, M.D., Professor of Anatomy, College of Physicians and Surgeons, Columbia University, New York, N. Y. Cloth. Pp. 136, with many illustrations. Price, $14.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1971.

Dynamic Studies with Radioisotopes in Medicine. Proceedings of the Symposium on Dynamic Studies with Radioisotopes in Clinical Medicine and Research organized by the International Atomic Energy Agency, and held in Rotterdam, Aug. 31–Sept. 4, 1970. Paper. Pp. 901, with some figures. Price, $24.00. International Atomic Energy Agency, Vienna. UNIPUB, P. O. Box 433, New York, N. Y. 10016, 1971.

Radiotomographic Anatomic Atlas. By Professor Dr. Hermann Schmidt, Chefarzt der Abteilung für Röntgendiagnostik im Allgemeinen Krankenhaus Hamburg-Altona; in collaboration with Professor Dr. K. Mörike, Anatomisches Institut der Universität Tübingen, Germany. Cloth. Pp. 223, with 194 plates. Price, $25.00. Hafner Publishing Company, Inc., 866 Third Avenue, New York, N. Y. 10022, 1970.

Principles of Chest X-Ray Diagnosis. Third edition. By George Simon, M.D., F.R.C.P., F.F.R., Lately Director, X-ray Department, Brompton Hospital, London; Radiologist, St. Bartholomew's Hospital, London; Demonstrator, Radiological Anatomy, St. Bartholomew's Hospital, London; Curator of the Radiological Museum and Teacher of Radiology, The Institute of Diseases of the Chest, University of London; Kodak Research Scholar, National Heart Hospital, London. Cloth. Pp. 282, with many figures. Price, $23.50. Appleton-Century-Crofts, 440 Park Avenue South, New York, N. Y. 10016, 1971.

Radiologic Atlas of Pulmonary Abnormalities in Children. By Edward B. Singleton, M.D., Director of Radiology, St. Luke's Episcopal Hospital, Texas Children's Hospital, and the

Texas Heart Institute; Clinical Professor of Radiology, Baylor University College of Medicine, Houston, Texas; and Milton L. Wagner, M.D., Associate Radiologist, St. Luke's Episcopal Hospital, Texas Children's Hospital, and the Texas Heart Institute; Clinical Assistant Professor of Radiology, Baylor University College of Medicine, Houston, Texas. Cloth. Pp. 251, with many figures. Price, $13.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1971.

CERVICAL SPONDYLOSIS: ITS EARLY DIAGNOSIS AND TREATMENT. Edited by Marcia Wilkinson, D.M., F.R.C.P., Consultant Neurologist, Elizabeth Garrett Anderson Hospital and the Hackney Group of Hospitals; Director, Regional Neurological Unit, Eastern Hospital, Hackney; and Director, City Migraine Clinic. Cloth. Pp. 182, with 112 illustrations. Price, $11.25. In the Western Hemisphere, W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1971.

CORONARY HEART DISEASE. International Symposium in Frankfurt, Jan. 22–24, 1970. Edited by Martin Kaltenbach and Paul Lichtlen. Paper. Pp. 271, with 138 figures and 57 tables. Price, DM 39.-. Georg Thieme Verlag, Stuttgart. In U.S.A. and Canada, Intercontinental Medical Book Corporation, New York, N. Y. 10016, 1971.

UNTERSUCHUNGEN ZUR STATIK UND DYNAMIK DER KINDLICHEN HALSWIRBELSÄULE: DER AUSSAGEWERT SEITLICHER RÖNTGENAUFNAHMEN. By Dr. med. Horst Markuske, Facharzt für Kinderheilkunde, Facharzt für Röntgendiagnostik und Facharzt für Sportmedizin, Lübbenau/Spreewald. Die Wirbelsäule in Forschung und Praxis, Band 50. Paper. Pp. 51, with some figures. Price, DM 18.-. Hippokrates Verlag, Stuttgart, 1971.

EFFETTI DELLE RADIAZIONI IONIZZANTI SUL CUORE (STUDIO CLINICO E SPERIMENTALE). By Gian Giacomo Ferri, and Umberto Dusi. Paper. Pp. 213, with many illustrations, Price, L. 5000. Edizioni Minerva Medica, Torino, Italy, 1969.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual meeting: Sheraton Hotel, Boston, Mass., September 28–October 1, 1971.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Jerome M. Vaeth, Saroni Tumor Institute, 1600 Divisadero St., San Francisco, Calif. 94115. Annual meeting: Boca Raton Hotel and Club, Boca Raton, Fla., May 14–19, 1972. Conjoint meeting with the James Ewing Society and the American Society for Head and Neck Surgery.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., November 28–December 3, 1971.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Bal Harbour, Florida, Americana Hotel, April 3–8, 1972.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: San Francisco, Calif., June 18–22, 1972.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Dallas, Tex., Dec. 6–10, 1971, Statler-Hilton Hotel; Washington, D.C., June 5–9, 1972, Washington-Hilton Hotel; and Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel.

Written examinations are scheduled in June of each year in 13 large centers, and applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1972 is September 30, 1971.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, Leonard Stanton, Hahnemann Medical College, 230 N. Broad St., Philadelphia, Pa. 19102. Annual meeting to be announced.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Carl R. Bogardus, Jr., University of Oklahoma Medical Center, Oklahoma City, Oklahoma 73104. Annual Meeting: Arizona Biltmore Hotel, Phoenix, Ariz., Oct. 29–31, 1971.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, F. J. Fry, M.Sc., Bioacoustics Lab., University of Illinois, Urbana, Ill.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary-Treasurer, Dr. Eugene V. Leslie, Edward J. Meyer Memorial Hospital, 462 Grider St., Buffalo, N. Y. 14215.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President, Dr. Juan Gómez López. Secretary of the Sociedad Española de Radiología, Dr. José Bonmati, Villanueva 11, Madrid 1, España.
Meeting: Madrid, Spain, Oct. 13–19, 1973.

TENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Manuel Viamonte, Jr., University of Miami School of Medicine, Jackson Memorial Hospital, Miami Fla. 33136.
President, Dr. Victor A. Marcial, Puerto Rico Nuclear Center, Caparra Heights Station, San Juan, Puerto Rico 00935.
Meeting: San Jeronimo-Hilton Hotel, San Juan, Puerto Rico, May 16–22, 1971.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colo. 80907.

SECOND CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY.
President, Professor Dr. J. R. von Ronnen, State University of Leiden, The Netherlands.
Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, The Netherlands. Congress Meeting: Amsterdam, The Netherlands, June 14–18, 1971.

FIRST ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia. Meeting: Melbourne, Australia, Nov. 22–26, 1971.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. William V. Weldon, Medical Arts Building, Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Irwin M. Freundlich, Department of Radiology, Thomas Jefferson University Hospital, Philadelphia, Penn. 19107.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4412 So. Lookout Rd., Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Byron G. Brogdon, Dept. of Radiol., Univ. of New Mexico School of Med., Albuquerque, N. M. 87106. Annual Meeting: Albuquerque, N. M., May 11–13, 1972.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Ernst Ruder, Dept. of Radiol., Crawford Long Memorial Hosp., Atlanta, Ga. 30308. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Lucien F. Burke, Maj, MC, Radiol. Service, US Army Gen. Hosp., Frankfurt, APO 09757. Next Meeting: West Berlin, Oct. 1–3, 1971; and Frankfurt, Nov. 12–14, 1971.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Kenneth B. Robinson, 301 E 75th St., Apt. 11-A, New York, N.Y. 10021. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Glen M. Ebersole, 405 Spring St., Jamestown, N.Y. 14701. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, Childrens Hospital of Los Angeles, P.O. Box 54700, Los Angeles, Calif. 90054.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P. O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 p.m.

Central New York Radiological Society
Secretary-Treasurer, Dr. David N. Cheris, Community General Hospital of Greater Syracuse, Broad Road, Syracuse, N. Y. 13215. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary-Treasurer, Dr. William T. Moss, 250 E. Superior St., Chicago, Ill. 60611. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 p.m. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Marvin L. Daves, Univ. of Colorado Medical Center, 4200 E. Ninth Ave., Denver, Colo. 80220. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Fred H. Dunn, 5940 Forest Park Rd., Suite 101, Dallas, Tex. 75235. Meets the 3rd Monday of every month at 6:30 p.m., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. John C. Osmer, 20103 Lake Chabot Rd., Castro Valley, Calif. 94546. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. Eberhard F. Besemann, Baroness Erlanger Hospital, Chattanooga, Tenn. 37403. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Arthur R. Miller, North Miami General Hospital, 1701 N.E. 127th St., North Miami, Fla. 33161. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. George J. Meyer, Holy Cross Hospital, Ft. Lauderdale, Fla., 33308. Meets monthly, third Wednesday at 8:00 p.m. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, Dr. Roland P. Ernst, 3720 Washington Ave., St. Louis, Mo. 63108.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ghim L. Yeoh, Prof. Center Bldg., 1481 S. King St., Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 p.m.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Stardust Hotel, Las Vegas, Nev., June 12–16, 1972.

Houston Radiological Society
Secretary, Dr. Milton L. Wagner, 6621 Fannin, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 p.m., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. Jack L. Melamed, Grant Hospital of Chicago, 551 Grant Pl., Chicago, Ill. 60614. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Bldg. Louisville, Ky. 40202. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 p.m.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Harold L. Atkins, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Harry T. Vanley, St. Mary's Long Beach Hospital, Long Beach, Calif. 90083. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. George Burke, 4000 North Charles St., Baltimore, Md. 21218.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Siadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Webster Riggs, Jr., The University of Tennessee College of Medicine, Department of Radiology, Walter F. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday of each month at John Gaston Hospital.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 p.m. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 p.m.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 p.m., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. James E. Bell, 8700 W. Wisconsin Ave., Milwaukee, Wis. 53213. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ottis G. Ball, 5356 Balmoral Drive, Jackson, Miss. 39211. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 p.m.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Arthur A. Porporis, 100 N. Euclid Ave., St. Louis, Mo. 63108.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101. Biennial Symposium at the Holiday Inn, Bozeman, Mont., July 28–30, 1971.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. N. Patrick Kenney, 3618 S. 114th Ave., Omaha, Nebr. 68144. Meets third Wednesday of each month at 6 p.m. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89502.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St., Brookline Mass. 02146. Meets Oct. 22, 1971, Nov. 12, 1971, and the third Friday of each month, Jan. through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 p.m.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary, George Farmlett, 33 Round Bay Rd., Keene, N. H. 03246. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Donald A. Wolfel, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New York Roentgen Society
Secretary-Treasurer, Dr. Samuel H. Madell, 1. E. 82nd St., New York, N. Y. 10028. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 p.m. Annual Spring Conference: Waldorf Astoria Hotel, New York. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 520 Franklin Ave., Garden City, N. Y. 11530.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. Thomas Lee Taylor, Department of Radiology, University of Florida, Gainesville, Fla. 32601. Meets quarterly in March, June, September and December.

Northeastern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N.Y. 12801. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiological Society
Secretary-Treasurer, Dr. Kevin Ryan, Woodland Medical group, Woodland, Calif. 95695. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Joseph Hanson, 1544 South Byrne Road, Toledo, Ohio 43614.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Edward I. Miller, 301 Newport Blvd., Newport Beach, Calif. 92660. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Gerald L. Warnock, 11699 N. E. Glisan St., Portland, Ore. 97220. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Oregon 97005. Annual Meeting: Empress Hotel, Victoria, B. C., Canada, May 12–14, 1972.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. C. Jules Rominger, Misericordia Hospital, 54th St. and Cedar Ave., Philadelphia, Pa. 19143. Meets first Thursday of each month at 5 p.m., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Donald E. Gunderson, 3553 Bayard Dr., Cincinnati, Ohio 45208. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
Secretary, Dr. Ken C. Davidson, St. Luke's Hospital of Kansas City, Kansas City, Mo. 84111. Meets 5 times a year on given dates.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of Louisiana, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, 1820 Octavia St., New Orleans, La. 70115. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07206. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. John J. O'Brien, 292 Merrymount Dr., Warwick, R.I. 02888.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Gladden V. Elliott, 5565 Grossmont Center Dr., Suite 1, La Mesa, Calif. 92041. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. M. Pinson Neal, Jr., Medical College of Virginia, 1200 E. Broad St., Richmond, Va. 23219. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, Rochester General Hospital, 1425 Portland Ave., Rochester, N. Y. 14621. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 19–21, 1971.

San Antonio-Civilian-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 111 Dallas St., San Antonio, Texas 78205. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, 20 Bridge Rd., Kentfield, Calif. 94904. Meets quarterly at various hospitals (contact Secretary).

Santa Clara County Radiological Society
Secretary, Dr. Raymond L. Schwinn, 480 Monterey Ave., Los Gatos, Calif. 95030. Meets monthly at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Phillip W. Voltz, Jr., 120 Medical Professional Bldg., San Antonio, Tex. 78212. Annual meeting: Hotel Fontainebleau, Miami Beach, Fla. Nov. 1–4, 1971.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles 27, Calif. Annual meeting: Sheraton Hotel, Boston, Mass., September 26–27, 1971.

Society of Nuclear Medicine
Secretary, Dr. James J. Smith, 140 E. 54th St., New York, N. Y. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. Emerson C. Curtis, University Dr., Menlo Park, Calif. 94025. Meets second Wednesday of each month.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Haakon O. Haugan, 718 Quincy St., Rapid City, S. D. 57701. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. Aaron G. Fingerhut, 1000 W. Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Sixteenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 28–30, 1972.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting at the Marriott Hotel, Houston, Tex., March 23–25, 1972.

The Fleischner Society
Secretary-Treasurer, Eric N. C. Milne, M.B., Medical Sciences Bldg., University of Toronto, Ontario, Canada.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. R. Newell Ford, St. Mark Hospital, 803 North 2nd West, Salt Lake City, Utah 84103. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. Edward A. Kupic, Mary Fletcher Hosp., Burlington, Vt. 05401.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. William E. Sheely, 1231 Kingston Ave., Alexandria, Va. 22302.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Leland L. Burnett, 5765 S. Eddy St., Seattle, Wash. 98118. Meets quarterly.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. J. Dennis Kugel, 510-517 Med. Arts Bldg. Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Edgar Palmer, 650 Main St., New Rochelle, N. Y. 10801. Meets on third Tuesday of January and October and on two other dates.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert E. Douglas, 1209 S. Commercial St., Neenah, Wis. 54956. Meets twice a year, May and September.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. J. D. Grant, Memorial Hosp., Sheridan, Wyo. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Julio Astacio, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Octavio Toussaint Goribar. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department oi radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 p.m. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Secretary, Dr. Pierre Archambault, Hôpital Charle Le Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics.
Honorary Secretary Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Canadian Association of Radiologists
Honorary Secretary, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 109, Que., Canada. Annual meeting: Toronto, Ontario, Canada, Jan. 17–21, 1972.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Guy Duckett, 1385 est, rue Jean Talon, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September, through May.

College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W., Australia.

South America

Asociación Argentina de Radiología
Secretary, Dr. Lidio G. Mosca, Avda. Fral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Ateneo de Radiologia
Secretary, Dr. Víctor A. Añaños, Instituto de Radiología, Urqiza 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 p.m. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Sociedad Argentina de Radiología
Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Cxa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigaderio

Secretary, Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Ricardo Cortez, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Raúl Fernández Angula, Calle 43 No. 44-70 Barranquilla, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 4569, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Julio Ormeño del Pino, Instituto de Radiología "Cayetano Heredia," Hospital Arzobispo Loayza, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41–110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de la Plata
Secretary. Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Miguel A. Sica, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Octavio Machado, Colegio de Médico del Dístrito Federal, Plaza de Bellas Artes, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Rontgen-Gesellschaft
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Permanent Secretary, Clément Fauré, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France.

Société Française d'Electroradiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord. Central Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française-de Neuroradiologie
Secretary General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting: Dec. 1971.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Rontgengesellschaft
President, Professor Dr. med. Hanno Poppe, Radiologische Univ.-Klinik, Gossler Str. 10, 34 Göttingen, Germany.

Società Italiana di Radiologia Medica e di Medicina Nucleare
Secretary, Dr. Ettore Conte, Ospedale Mauriziano Torino, Italy. Meets annually.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. C. B. A. J. Puijlaert, Prof. Dondersstraat 73, Tilburg, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. D. José Bonnati, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Schweizerische Gesellschaft Für Radiologie und Nuklearmedizin (Société Suisse de Radiologie et de Médecine Nucléaire)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month. Second Iranian National Congress of Radiology, Teheran, Iran, May 6–11, 1972. Deadline for acceptance of papers, Feb. 20, 1972.

Philippine College of Radiology
Secretary-Treasurer, Dr. Edmundo V. Villacorta, Box 1284, Commercial Center, Makati, Rizal D-708, Philippines. Meets fortnightly on Fridays. Annual Meeting: Jan. 1972.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan,Ibadan, Nigeria.

South African International radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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RHO, YONG-MYUN. An unusual complication of gastric ulcer. *Canad. M. A. J.*, April, 1971, *104*, 704–706. (From: Department of Pathology, St. John's General Hospital, St. John's, Newfoundland, Canada.)

The author reports a case of a perforation of a gastric ulcer into the left pleural cavity, undiagnosed prior to death.

A 52 year old male had sudden onset of left chest pain, dyspnea, cough and fever. The patient was hospitalized with the disclosure of a left hydropneumothorax. He was treated with antibiotics and underwater drainage of the left chest. Examination of the pleural fluid and bronchial washings failed to show evidence of malignancy. On the eighth hospital day, the drainage tube was inadvertently pulled out but was reinserted. A chest roentgenogram at that time disclosed expansion of the upper portion of the left lung, while a portion was obscured by fluid in the pleural cavity. There was considerable subcutaneous emphysema over the upper chest and neck. On the tenth hospital day, the patient expired after a short period of severe respiratory distress and cyanosis.

At autopsy, the source of the fluid and air in the left chest was traced to a hole in the left leaf of the diaphragm, 4 cm. to the left of a lateral margin of the esophageal hiatus. This opening measured 2.1 × 1.5 cm. It was identified as the outlet of a perforated gastric ulcer which was firmly adherent to the undersurface of the diaphragm. Microscopically, the wall of the perforation was made up of dense fibrous granulation tissue extending through the stomach and the diaphragm. There was no evidence of malignancy. —*Saul Heiser, M.D.*

WRIGHT, F. W., and MATTHEWS, J. M. Hemophilic pseudotumor of the stomach. *Radiology*, March, 1971, *98*, 547–549. (From: Department of Radiology, Churchill Hospital, Oxford, England.)

One of the complications of a hemorrhagic diathesis with anticoagulant therapy is intramural intestinal hemorrhage.

The authors report 2 patients with a bleeding diathesis who had bleeding into the wall of the stomach, causing difficulty in initial diagnosis. In one case, a possible antral carcinoma was initially considered, while in the other case the mass was large and easily palpable so that an abdominal wall hematoma was clinically thought to be likely. In both cases, the condition subsided under appropriate antihemophilic treatment.

Many of these patients demonstrate a characteristic roentgen pattern and recognition of this pattern and differentiation from other discrete lesions of the intestinal tract are important, since operation is unnecessary and the condition resolves without medical treatment. A solitary hematoma might be revealed by its increased density or by displacement of loops of the small or large bowel, whereas, if the hemorrhage is confined to the bowel wall, varying degrees of paralytic ileus, narrowing of the bowel lumen, or localized filling defects producing marked scalloping of the bowel may be seen. A "palisade" effect may also be seen on plain roentgenograms or contrast studies.

A review of the literature revealed only 2 other reports of bleeding into the stomach wall. One report was of an 8 year old hemophilic boy and the other concerned 2 cases without a bleeding diathesis.

The possibility of neoplasms developing in hemophilics must not be discounted lightly, as several examples of gastrointestinal and other tumors have been encountered.—*F. R. Jenkins, Jr., M.D.*

IBRAHIM, KAMAL ABDALLA. True gastro-duodenal diverticula: review of gastro-duodenal diverticula and report of eight cases. *Kasr-el-Aini J. Surg.*, Oct., 1970, *11*, 299–312. (From: Department of Surgery, Ras-El-Tin Hospital, Alexandria [Egypt], U.A.R.)

Gastric diverticula are uncommon but duodenal diverticula are very common and are seen routinely in about 2 per cent of patients having upper gastrointestinal examination.

The author states that duodenal diverticula may be congenital, acquired, or false. It is frequently not possible to decide on the basis of roentgenographic examination which type of diverticulum is being observed.

During a 5 year period between 1963 and 1968, the author operated on 8 cases of gastroduodenal diverticula in patients suffering from dyspepsia. Two of the cases were gastric in origin and 6 were duodenal in origin. The results of surgical treatment were uniformly excellent, but the author points out that these patients were carefully selected and all had had long periods of medical management which had failed to cure the patient's symptoms.

Emphasis is placed on the fact that only those patients with symptoms not relieved by medical management should be subjected to surgical treatment. —*Richard E. Kinzer, M.D.*

HUMPHRY, A. Intestinal obstruction due to abnormal duodenal fixation in infants: two case reports. *J. Canad. A. Radiologists*, Dec., 1970, *21*, 251–256. (From: Department of Radiology, Hospital for Sick Children, Toronto, Ontario, Canada.)

Errors in development of the intestine may precipitate a sudden obstruction in the gastrointestinal tract at some time following the birth of the infant.

The authors report 2 cases of intestinal obstruction as the result of malformations which were proven surgically.

In their discussion an excellent resumé is given of the series of rotations of the intestine to reach the adult position.

The text is supported by excellent diagrams and reproductions of roentgenograms.—*David C. Alftine, M.D.*

Fraser, G. M., MacFarlane, P. S., and Main, R. G. Coarse duodenal mucosal folds in patients with dyspepsia and a high gastric acid output. *Clin. Radiol.*, Jan., 1971, *22*, 78–82. (From: Departments of Radiodiagnosis, Pathology and Surgery, Falkirk and District Royal Infirmary, Scotland.)

"Coarse mucosal folds" is a common terminology used to describe a cobblestone mucosal pattern in the duodenal cap and frequently extending into the second part of the duodenum. The cause of this roentgenographic appearance has been defined as being due to edema of the mucosa and sometimes as due to adenomas of Brunner's glands.

The authors believe that the cobblestone appearance of the mucosa is rarely due to Brunner's glands hypertrophy and is probably the result of excessive activity of the muscularis. They report 6 patients with dyspepsia of sufficient severity to warrant surgical treatment. All of these patients had barium meal examinations showing coarse duodenal mucosal folds without evidence of ulceration. A biopsy was taken from the first part of the duodenum in 5 of the cases operated.

The mean output in the cases reported was 52.7 mEq./hr. Others have found that the mean acid output in men with coarse duodenal mucosal folds was 44.3 mEq./hr.; in men with duodenal ulcer 41.5 mEq./hr., and in normal men, only 25.3 mEq./hr. Thus, coarse mucosal folds in the duodenum seen at barium meal examination strongly suggest high gastric acid output; the roentgenologic appearances are also probably partially due to excessive activity of the muscularis rather than to mucosal edema.

These patients should be treated as if they had active duodenal ulceration. In fact, associated ulceration unsuspected at roentgenographic examination is not an uncommon finding at surgery.

The histology of the biopsy specimens showed extensive gastric type epithelium on the surface of the villi in many of the cases reported.—*Richard E. Kinzer, M.D.*

Rösch, Josef, Dotter, Charles T., and Rose, Robert W. Selective arterial infusions of vasoconstrictors in acute gastrointestinal bleeding. *Radiology*, April, 1971, *99*, 27–36. (From: Stella and Charles Guttman Laboratory for Research, University of Oregon Medical School, Portland, Ore.)

The vascular catheter recently has become important in the nonsurgical treatment of acute gastrointestinal bleeding. Two basic techniques have been developed. In one, a balloon catheter is used for obstruction of an affected vessel and temporary control of bleeding prior to surgery. In the other method, the selective intraarterial infusion of vasoconstrictors is used to achieve control of bleeding. Recent reports indicate the arrest of variceal bleeding by the infusion of pitressin into the superior mesenteric artery, and the definitive control of arterial gastrointestinal bleeding treated by selective arterial infusion of epinephrine alone or in combination with propranolol.

The authors report their technique and experience with treatment of 21 patients with acute gastrointestinal bleeding, 16 arterial and 5 with esophageal varices.

Of the patients with arterial bleeding, emergency diagnostic angiography was performed in all, with usually selective celiac catheterization and often superior mesenteric angiography. Superior and inferior mesenteric angiograms were obtained if lower gastrointestinal bleeding was clinically evident. Selective vasoconstrictor infusions were given only when a positive angiographic diagnosis of active bleeding could be made by the demonstration of extravasated contrast material into the lumen of the bowel. The excellent data on these patients are arranged in tabular form, and a review of this material is encouraged for the interested reader. The selection of the artery for infusion depends on the arterial supply of the bleeding point. Epinephrine alone is infused into the celiac artery or its branches and is sufficient to produce hemostasis. However, when the mesenteric arteries are to be perfused, a preinjection of 3 to 5 mg. of propranolol, a beta-adrenergic blocking agent, has been routine since beta stimulation by epinephrine alone can cause significant vasodilatation in the mesenteric circulation. In general, 12 to 20 μg./min. of epinephrine is infused into the celiac, hepatic, or superior mesenteric arteries, while 6 to 10 μg./min. suffices for inferior mesenteric, left gastric or gastroduodenal artery infusions. In the first 5 patients, 4 to 6 minutes of infusion was given but in subsequent patients, 20 minute infusions were given. In a few difficult cases the infusions were prolonged to 60 minutes. Following infusion, those catheters in the celiac or superior mesenteric artery were left in place 24 to 48 hours for possible use in the event of further bleeding. It is noteworthy that in 11 of 16 patients the infusion leads to immediate, complete and permanent cessation of arterial bleeding in the gastrointestinal tract.

Five patients were treated for bleeding from esophageal varices. In each instance, diagnostic angiography with selective celiac and superior mesenteric injection was first performed. The diagnosis of the variceal bleeding was based on the presence of clinically active bleeding, the absence of angiographically evident arterial bleeding, and angiographic evidence of portal hypertension with esophageal or gastric varices. Superior mesenteric artery infusions of pitressin, a peripheral vasoconstrictor, were given to all 5 patients. At this time, the control of variceal bleeding is sought through infusion of the superior mesenteric artery since it carries over half of the blood which traverses the portal circulation. Constriction of this vessel, especially in portal hypertension, can bring about a considerable fall in portal pressure. The dosage of pitressin varies from 0.1 to 0.3 units per minute for 2 to 6 hours. Three of the 5 patients had uncomplicated cirrhosis and, in each on these, pitressin infusions were associated with complete cessation of variceal hemorrhage.

On the basis of experience to date, the authors believe that selective arterial vasoconstrictive infusion offers encouraging means for the suppression of acute gastrointestinal bleeding, and thus constitutes a promising new therapeutic method. The local infusion of vasoconstrictive drugs can substantially reduce regional blood flow, thereby making possible stable clot formation and permanent occlusion of the bleeding vessel. Vessels capable of contraction and an adequate coagulation mechanism are prerequisites to successful treatment. When these were present, bleeding was promptly, completely, and permanently controlled, usually by a single infusion.—*Edward B. Best, M.D.*

BAUM, STANLEY, and NUSBAUM, MOREYE. The control of gastrointestinal hemorrhage by selective mesenteric arterial infusion of vasopressin. *Radiology*, March, 1971, *98*, 497–505. (From: Departments of Radiology, and Surgery, Graduate Hospital, University of Pennsylvania, Philadelphia, Pa.)

The authors studied 48 patients, over the past 2 years, with selective mesenteric arterial infusion of vasopressin for the control of gastrointestinal hemorrhage.

In the first group of 28 patients, who were actively bleeding from varices secondary to portal hypertension, vasopressin infusion into the superior mesenteric artery adequately controlled the bleeding in 27 cases. Following arteriography the catheter was placed in the superior mesenteric artery with continuous infusion of vasopressin at the rate of 0.2 pressor units per milliliter per minute, with the time of infusion ranging from 3 to 14 days.

In a second group of 14 patients selective superior mesenteric arterial infusion of vasopressin during portosystemic shunt surgery resulted in a significant collapse of the collateral pathways, facilitating surgical exposure and dissection, and decreasing operative time and blood loss.

In a third group of 6 patients with massive gastrointestinal bleeding, the bleeding was adequately controlled by selective infusion of vasopressin into the left gastric artery in 5 patients and into the superior mesenteric artery in the 6th patient.

The indications, technique, and results of vasopressin infusions are described and discussed in detail.—*Wm. L. Graham, M.D.*

GENITOURINARY SYSTEM

THOMPSON, DENNIS P., MOORE, THOMAS C., and FINGERHUT, AARON G. A method for high resolution renal and splenic microangiography. *Surg., Gynec. & Obst.*, Jan., 1971, *132*, 101–108. (From: Surgical Research Laboratory, UCLA-Harbor General Hospital, Torrance, and Departments of Surgery and Radiology, University of California, Los Angeles, Calif.)

A new microangiographic technique was evaluated in 80 canine kidneys, and in 5 canine spleens. The freshly excised organ was immediately perfused with heparinized saline at a constant pressure of 200 mm. Hg. The renal vein perfusate was usually clear of blood after about 300–500 ml. of solution. A barium suspension of 1 part barosperse or micropaque to 10 parts normal saline solution was then perfused at the same constant pressure, until no more suspension could be given without increasing the pressure. Serial 1 mm. slices (5 mm. for the spleens) of the barium-infused kidneys were cut immediately after infusion, and roentgenograms exposed on a high resolution glass plate or high resolution emulsion industrial x-ray film. Using this technique, consistently good renal microangiograms can be obtained with excellent visualization of interlobular arteries, afferent arterioles, and glomerular tufts.

The experiments included normal kidneys as well as first and second set allotransplanted kidneys with and without immunosuppression. The transplanted kidneys showed areas of fairly normal vasculature immediately adjacent to areas virtually devoid of glomeruli. Peripheral glomeruli are lost first and the more proximal ones last.

This new technique provides a parameter which can be applied to the study of the rejection phenomena, and when correlated with light and electron microscopy and renal function studies, may help to resolve some of the unanswered questions concerning the nature of rejection and the suitability of various perfusion and storage methods.—*Mark D. Reiss, M.D.*

SKELETAL SYSTEM

GOEMINNE, L., and DUJARDIN, L. Congenital coxa vara, patella aplasia and tarsal synostosis: a new inherited syndrome. *Acta genet. med. et gemmellol.*, Oct., 1970, *19*, 534–545. (From: Medical Clinic and Department of Human Genetics, Ophthalmologic Clinic, University of Ghent, Belgium.)

A 44 year old German female is reported with roentgenographic findings of congenital coxa vara, patellar aplasia, and tarsal synostosis. There were ancillary findings of bilateral hypoplasia of the descending parts of the pubic arches, shallow acetabula but no hip dislocation, mild bilateral shortening of the femora and only 4 metatarsal bones bilaterally, with bifidity of the fourth metatarsal on the right foot. The synostosis was between the talus and calcaneum bilaterally and also between the cuneiform bones. The colon was described as involved by severe colitis, without further amplification.

Clinically, the mobility of the joints was normal; there was no nail dystrophy; and there were normal tendinous reflexes. In general there was a bizarre dysmorphic habitus. She was of short stature. Bilaterally the femora seemed to be shortened, and there was absence of patellae associated with flat feet and bilateral hallux valgus. There were 4 toes on the left foot and 5 toes on the right foot. The patient walked with difficulty. The shoulders and the upper extremities were strictly normal. The somatic features were: height 144 cm.; weight 66 kg.; span 170 cm.; vertex to pubis 92 cm.; and pubis to ground 52 cm.

The patient has an 18 year old daughter with bilateral patellar aplasia and essentially the same bilateral pelvic and hip dysplasia including absent ischiopubic synostoses bilaterally, subluxation of the right hip, and bilateral severe coxa vara. There is no tarsal synostosis. A 4 year old son was reported as having no detectable patellar ossification as yet. Contact with other family members was lost during World War II.

There is a discussion of these various congenital abnormalities as they occur as isolated defects or in combination with other congenital abnormalities not found in these cases.

The authors conclude that the nosologic association of these multiple defects has never been published so far, and they suggest that this association is probably inherited as an incomplete dominant trait which apparently occurred by new mutation.—*Donald M. Monson, M.D.*

Callaghan, K. A. Thanatophoric dwarfism: a case report. *Australasian Radiol.*, Nov., 1970, *14*, 435–437. (Address: Paediatric [Neonatal] Registrar, The Queen Elizabeth Hospital, Woodville, South Australia.)

This is a case report of thanatophoric dwarfism.

A point is made of the possibility of diagnosing it *in utero*.

The need for genetic counseling of this fatal form of achondroplasia is stressed.—*Victor G. Mikity, M.D.*

BLOOD AND LYMPH SYSTEM

Patil, Khusal D., and Williams, Kenneth Lloyd. Thermographic study of heat flow in the detection of incompetent perforating veins. *Surg., Gynec. & Obst.*, March, 1971, *132*, 396–402. (From: Department of Measurement and Clinical Research, St. Martin's Hospital, Bath, Somerset, England.)

The successful treatment of varicose veins by either operation or injections depends upon accurate localization of incompetent perforating veins, found in about 85 per cent of the patients. The preoperative detection of all incompetent perforating veins is, however, difficult.

Clinical methods detect only 50 to 60 per cent of incompetent perforating veins and give a considerable number of false-positive results. With the use of venography, an accuracy rate of 85 to 90 per cent in the diagnosis of incompetent perforating veins has been reported. This procedure is, however, costly. Difficulties are experienced in correlating roentgenologic findings with actual sites of incompetent perforating veins. It was recently reported that skin temperature is higher at the site of incompetent perforating veins than over surrounding areas.

The rise in skin temperature overlying the site of an incompetent perforating vein is caused by the retrograde flow of warm blood from the deep veins into the superficial veins. To test the validity of this concept, a quantitative study of skin temperature changes at the site of incompetent perforating veins was made. The authors report the results of this investigation.

The thermal scanner described by Griffiths and Lawson* was equipped with line scan facilities so that a thermal picture and a line scan could be obtained simultaneously.

Initial diagnosis of an incompetent perforating vein was made clinically as well as by the thermographic demonstration of a hot spot.

To exclude heat flow through collateral venous circulation, the superficial veins of the leg were occluded by tourniquets just below and above the site of the perforating vein. From the line scans, the degree and local extent of skin temperature rise caused by local heat flow were determined.

During exercise of the calf muscles, rapid rewarming of the skin overlying incompetent perforating veins was observed. The rate of temperature rise varied from patient to patient. The total change in skin temperature ranged from 1.5 to 6°C. at the end of 60 seconds. Temperature changes were directly related to the size of the perforating veins and the degree of incompetence. Peak of temperature rise was always at the site of the perforating vein.

The reappearance of a hot spot in a cooled and elevated leg with occlusion of superficial collateral veins distally and proximally forms a useful thermographic criterion for the diagnosis of incompetent perforating veins.

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* Griffiths, D. W., and Lawson, W. D. Infrared detectors and parameters for thermograph camera design. *Phys. Med. Biol.*, 1968, *13*, 470.

Fifty-five patients, 80 limbs, with varicose veins were studied by both clinical and thermographic methods. Of the 55 patients, 41 were women and 14 men. Their ages ranged from 20 to 74 years. All patients were treated by operation.

Incompetent perforating veins were diagnosed in 51 of 55 patients. In the remaining 4 patients, no incompetent perforating veins were detected clinically or thermographically, the main lesion being incompetence of the long saphenous vein.

Fifty-five (56 per cent) of the 98 sites diagnosed clinically and 96 (85 per cent) of the 113 sites diagnosed thermographically yielded incompetent perforating veins at operation. Of the total 101 incompetent perforating veins found, the clinical method detected 55 (54.5 per cent), missed 46 (45.5 per cent) and gave false-positive results in 43 instances (44 per cent). Thermography, however, detected 96 (95 per cent), missed 5 (5 per cent), and contributed 17 (15 per cent) false-positive results.

Clinical false-positive results were caused by fascial defects, large varices, and branching sites of superficial varices.

The thermographic method is relatively easy to perform, is innocuous and gives an objective assessment of a functional derangement.—*Stephen N. Tager, M.D.*

RADIATION THERAPY

PROUT, GEORGE R., JR., SLACK, NELSON H., and BROSS, IRWIN D. J. Preoperative irradiation as an adjuvant in the surgical management of invasive bladder carcinoma. *J. Urol.*, Feb., 1971, *105*, 223–231. (From: Urological Service, Massachusetts General Hospital and Harvard Medical School, Boston, Mass.; and Roswell Park Memorial Institute, Buffalo, N. Y.)

The authors report the progress of the Urological Cancer Research Group, describing observations pertinent to the question of preoperative irradiation as an adjunct in the surgical management of patients with invasive bladder cancer. Previous reports from the authors included details on the group's formation, its experiences and an analysis of the effectiveness of 5-FU as definitive therapy in the treatment of advanced bladder cancer.

The present report deals with the 5 year status of a cooperative program designed to determine the efficacy of preoperative radiation therapy when used as an adjunct to surgery in the management of invasive bladder cancer. There were 427 patients entered, 242 currently eligible and 185 patients ineligible. The ineligible category included 93 patients who were excluded from the study and 92 called protocol deviates because, while they did not finish the study, they proceeded far enough to provide data helpful in answering certain questions relative to the efficacy of preoperative irradiation of bladder carcinoma.

The preoperative radiation therapy was effective in destroying the bladder tumor in about one-third of the surgical specimens. When irradiation was not used, the diagnostic procedure removed the tumor 7 per cent of the time.

Of the nonlethal complications the only statistically significant increase was found in wound infections. Lethal complications occurred without respect to treatment group. Estimates of clinical staging demonstrated no close correlation with the true stage and this defect makes accurate evaluation of the effect of preoperative irradiation difficult. The life table curve for all eligible patients demonstrated rapid survival loss in the first 15 to 18 months postoperatively with relatively few deaths occurring after 18 months. Evaluation of the data demonstrated that patients irradiated or not, and with or without tumor in the surgical specimen, suggested some initial advantage in survival for those whose tumors were eradicated by preoperative irradiation. Patients with tumor eradicated by the diagnostic procedure (nonirradiated, no tumor) had a good prognosis with 100 per cent survival at 12 months, but experienced rapid loss thereafter to a survival probability similar to the other groups (approximately .40) at 30 months. Ninety-five per cent confidence limits for the different forms of therapy show overlap, supporting the conclusion that no regimen is better than another. Metastases found in patients who were treated according to the protocol requirements were widespread, showing no predilection for local recurrence and occurred without regard to whether the patient had received preoperative irradiation.—*Luther W. Brady, M.D.*

LEDLIE, E. M., MYNORS, L. S., DRAPER, G. J., and GORBACH, P. D. Natural history and treatment of Wilms's tumour: an analysis of 335 cases occurring in England and Wales 1962–6. *Brit. M. J.*, Oct., 1970, *4*, 195–200. (From: Department of Social Medicine, 8 Keble Road, Oxford University, Oxford, England.)

Hospital records and other data relating to Wilms' tumor were analyzed to elucidate both the natural history of the disease and the effects of treatment, with special reference to actinomycin D. The study represents 335 cases of Wilms' tumor and is thought to comprise all the children in England and Wales under the age of 10 years who were diagnosed during the 5 year period 1962–66.

Half of the tumors presented before the age of 3 years, the peak incidence being at 18 months. The left-sided tumors were significantly more common than right-sided tumors. Congenital tumors occurred in 10 children, 6 of whom have survived for 3 years or more without developing signs of further disease.

In most cases, an abdominal mass was palpable and treatment was promptly instituted. Abdominal pain was present in one-third of the cases. Hematuria and general symptoms (pyrexia, lassitude, and anorexia) each occurred in about one-quarter of the cases. Eighteen children gave a history of abdominal symptoms following an injury, 8 were admitted as acute abdominal emergencies, and 5 had no symptoms at all, the tumor being discovered by the physician examining the child for an unconnected symptom.

Prognosis was related to the stage of the disease at the initial operation and to the occurrence of metastasis. The 3 year survival rate for cases having a nephrectomy was 35 per cent.

A recurrence was observed in 156 cases, of whom 141 have died. The 3 year survival rate for these cases was 12 per cent. No recurrence has been observed in 76 cases, all of whom have survived.

The pattern of recurrence times strongly suggests that beyond 2 years the chance of recurrence is very small; 107 of the 335 cases survived 3 years after diagnosis. About two-thirds of Stage A cases and a few Stage C cases survived; older children have a worse prognosis than younger children. Only a small proportion of cases with metastases survive, and this is true whether the metastases were discovered at the time of operation or later.

Nephrectomy was performed on 304 children (90 per cent of the cases) and the over-all survival rate was 35 per cent. No patient who did not have a nephrectomy survived.

Assessment of the effect of adding radiotherapy and chemotherapy to nephrectomy is difficult, since the groups of cases receiving different treatment combinations are not comparable.

The effects of radiotherapy and chemotherapy are considered in detail. Very little improvement in survival rates can be ascribed to actinomycin D. The reason for this is the variation found in other reports of selected cases, possibly the addition of other components of treatment, and differences in dose regimens.

The authors state that the evidence in favor of actinomycin D is such that it seems ethically impossible to withhold it at this stage. They suggest further trials to evaluate various different drug schedules with a sufficiently long follow-up to ensure that recurrences are being prevented and not simply postponed.—*James F. Martin, M.D.*

Lipton, Allan, and Lee, Burton J. Prognosis of Stage I lymphosarcoma and reticulum-cell sarcoma. *New England J. Med.*, Feb., 1971, *284*, 230–233. (Address: Dr. A. Lipton, Salk Institute, P. O. Box 1809, San Diego, Calif. 92112.)

Lymphosarcoma and reticulum cell sarcoma may be clinically localized at the time of first diagnosis. When this presentation is the case, the prognosis for the future is markedly improved. Various investigators have reported a highly favorable outcome in situations such as this.

The authors report their investigation of the course of patients with lymph nodal or extranodal Stage I lymphosarcoma and reticulum cell sarcoma with negative lymphangiography.

Eleven patients with Stage I (negative lymphangiography) lymphosarcoma and 15 patients with Stage I reticulum cell sarcoma were followed for at least 2 years. There were 3 deaths and 6 recurrences, all within 32 months of diagnosis. There were 92 per cent alive at 2 years and 85 per cent at 4 years. Early bone marrow invasion was seen only in the 3 patients who died. Six patients survived for a mean of over 111 months with lymph nodal and extranodal recurrences. There were 2 deaths and 6 recurrences in 19 patients (42 per cent) receiving less than 3,500 r and only 1 death and no recurrences in 7 patients receiving at least 3,500 r or more to the primary tumor site.

Radiotherapy appears to offer an excellent chance for cure when the dose delivered to the tumor is greater than 3,500 rads with a supervoltage device. The treatment field should include the entire primary site as well as a generous margin of normal tissue surrounding it. There is no evidence at this time to suggest that contiguous lymph node chains should be treated. Systemic chemotherapy and radical radiotherapy should not be employed in the management of these diseases at this stage.—*Luther W. Brady, M.D.*

Rudnick, P., and Odell, W. D. In search of a cancer. *New England J. Med.*, Feb., 1971, *284*, 405–408. (Address: Dr. W. D. Odell, Harbor General Hospital, 1000 West Carson Street B5, Torrance, Calif. 90509.)

There is increasing evidence that a large variety of neoplasms elaborate polypeptides with hormonal properties. Such neoplasms are of particular importance to the oncologist because the produced hormone may serve as a label of the cancer cell. The hormonal label has been useful to indicate the presence of the cancer itself, aiding in diagnosis, and in following response to treatment.

The authors report their experience with a young man in whom newer diagnostic techniques demonstrated the positive presence of a neoplasm and in whom an extensive search was conducted for 1 year prior to diagnosis without discovery of the neoplasm. The patient ultimately died of widespread metastatic disease.

the vessel and with a tendency to accumulate in rings.

Correlation of these different types of calcification with cerebral physiologic functions reveals that the group with intimal calcifications had a significantly lower cerebral blood flow than the group with medial calcification. The cerebral vascular resistance of the intimal group was greater than that of the medial group, but not significantly so, and there was no significant difference between the group with absence of calcification and the groups with intimal or medial types of calcification.

In view of these findings, the authors feel that the distinction of the type of carotid siphon calcification should be attempted roentgenologically as it may prove useful for the clinical appraisal of cerebrovascular syndromes.—*Donald N. Dysart, M.D.*

NECK AND CHEST

Mejía, Jorge Ceballos. (Colombia). Aspectos radiológicos de la neumonia estafilococcica en le niño: revision de 39 casos. (Radiologic aspects of staphylococcal pneumonia in children: review of 39 cases.) *Rev. Interam. Radiol.*, 1970, *5*, 31–38.

Staphylococcal pneumonia is predominantly a pediatric disease. It can occur as a primary process (originating in the upper respiratory tract); it can complicate a viral process; or be secondary to distant septic foci (*Staphylococcus septicemia*).

The author reviews 39 cases treated at the Misericordia Children's Hospital: 23 of the patients were less than 2 years old (16 were less than 1 year old); and 16 were older than 2 years (up to 12 years). There were 29 males and 10 females. Nine patients died; no age preponderance was noted.

Thirty-two cases were bacteriologically proven; in the other 7 cases the diagnosis was based on the radiologic and clinical picture.

In 14 cases only the respiratory system was involved (primary cases); in the others the pneumonia was considered to be secondary to septicemia (8 cases), soft tissue infections (7 cases), osteomyelitis (5 cases), and meningitis (5 cases).

The radiologic findings were as follows: (a) lobar consolidation- 8 cases; (b) bronchopneumonic infiltrations- 30 cases (frequently of the macronodular type in the secondary cases); (c) pneumatoceles- 30 cases; (d) true abscesses- 12 cases; and (e) pleural changes- 24 cases (11 showed small pleural effusions, 5 had large pleural effusions, 8 had hydropneumothorax, and 1 case had pleural change without pulmonary findings).

In 30 cases the pulmonary changes were bilateral —an interesting finding as most authors affirm that the involvement is usually unilateral.

Noted was the fact that the lung lesions changed relatively fast (clearing in one site and appearing in another; rapidly developing pneumatoceles with rapid size changes). The pneumatoceles can persist after the patient is clinically cured.—*Julian B. Alvarez, M.D.*

Phibbs, Brendan P., Sundin, Robert E., and Mitchell, Roger S. Silicosis in Wyoming bentonite workers. *Am. Rev. Resp. Dis.*, Jan., 1971, *103*, 1–17. (Address: Dr. Roger S. Mitchell, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver, Colo. 80220.)

"Bentonite" is a term applied to a group of clays mined in Wyoming and in several other parts of this country. These clays, also known as "mintmorrilonite" and "beidelite" are formed from devitrification of volcanic ashes deposited in water and altered in place.

The free silica content of bentonitic clays ranges from 1 to slightly more than 24 per cent. Bentonite is not a true mineral and has, therefore, no precise chemical formula.

Bentonite clays are useful because of the great water-retaining properties of the substance. Common usages include filler substances for crayons, the manufacture of concrete and a lubricant in oil-well drilling.

The mining of bentonite clays is performed in open pits and the dust is usually blown away by air currents. The milling process, however, produces very large quantities of dust indoors.

Dust analysis made in bentonite milling plants revealed dangerous concentrations of silica various times during the past 13 years. Silicotuberculosis developed in 4 patients, and a totally disabling or fatal disease occurred in at least 4 workers after periods of exposure of from 6 to 14 years.

Bentonite dust, once believed to be harmless, must now be added to the list of hazardous dusts because of its content of free crystalline silica.

The roentgenographic findings in the chest of patients developing silicosis due to bentonite exposure are identical to the roentgenographic appearance of silicosis contacted in other forms of exposure.—*John H. Harris, M.D.*

Remy, J. Thymus et myasthenie: aspects radiologiques. (Thymus and myasthenia: radiological aspects.) *Lille méd.*, Nov., 1970, *15*, 1308–1309.

Fifteen per cent of patients with myasthenia have thymomas (lymphoepitheliomas), 80 per cent of which are encapsulated and 35 per cent quite small, down to 5 grams. Many of the tumors are missed in frontal views, and tomography is useless in the absence of gaseous contrast. Pneumomediastinum, however, is likely to outline the tumor wall, and to permit identification of separating planes of tissue although a large tumor by its sheer volume may prevent the migration of air.

Caval phlebography may be of use even without compression of the superior vena cava, since a compressing thymoma may invade the left brachiocephalic trunk without any signs suggesting this complication. Thymic arteriography can be done by injection of subclavian, internal mammary or inferior thyroid vessels, but carries some hazard; preferably it is done bilaterally. Still better is thymic phlebography since the thymic veins unite in 1 or 2 collectors terminating at the inferior border of the left brachiocephalic trunk. This procedure is better tolerated than pneumomediastinum, does not add to respiratory embarrassment, and permits differentiation between tumor and simple hypertrophy.—*Frank A. Riebel, M.D.*

ABDOMEN

Moir, J. D. Combined duplication of the esophagus and stomach. *J. Canad. A. Radiologists*, Dec., 1970, *21*, 257–262. (From: Saint John General Hospital, Saint John, New Brunswick, Canada.)

The author reports a case in a 9 day old infant who was found to have complete duplication of the esophagus and the stomach.

The case is unusual in that only one esophagus could be filled through the stomach, while the other esophagus was filled retrograde from the stomach following surgery.

An excellent discussion of the complications is given.—*David C. Alftine, M.D.*

Herrera, Carlos Alberto. Radiología de la regíon esofagocardiotuberositaria normal y patológica. (Normal and pathologic radiology of the esophago-cardio-tuberosity area.) *Rev Argentina Radiol.*, Sept.–Dec., 1970, *33*, 247–262. (Address: Profesor Titular de Radiología de la Facultad de Medicina de Tucumán, Tucumán, Argentina.)

Anatomically this area has been divided into several segments.

1. *Epiphrenic segment.* This segment measures about 3–4 cm.; it is limited inferiorly by the hiatus and superiorly by a primary peristaltic wave provoked by deep inspiration or the Valsalva maneuver. This segment is also known as "the ampulla of Hasse-Strecker," or "the lower functional esophageal sphincter of Lerche."

2. *Intrahiatal segment.* It measures about ½ cm., there being a slight narrowing at this level.

3. *Abdominal segment.* It measures 2–4 cm. Its lateral (left) margin forms with the medial margin of the gastric fundus an acute angle ("the angle of His"). Within the lumen of the stomach, there is a mucosal valve ("the valve of Gubaroff") which corresponds with this external angle. This valve plays an important part in gastroesophageal continence.

4. *Cardiac segment.* This is an oval orifice 2 cm. in longest diameter; in its normal resting state it is closed. A true sphincter has not been demonstrated. It is the diffuse contraction of the abdominal segment that acts as a sphincter.

5. *Major tuberosity (gastric fundus).* This corresponds to the area above a transverse plane through the cardia. The average length is 5 cm.

The normal radiographic appearances are:

1. *Epiphrenic segment.* The ampulla is seen as a transitory smooth oval dilatation appearing during deep inspiration; if this is maintained, the ampulla empties proximally after the peristaltic wave above it subsides.

2. *Intrahiatal segment.* Its projection varies according to position of observation. As seen frontally and in a slight right anterior oblique (RAO) position, it projects above the left diaphragm. In a sharp RAO it projects at or below the diaphragm.

3. *Abdominal segment.* Its length is variable, sometimes it is indistinguishable.

4. *Cardiac segment.* If seen "en face" and opened, it presents as a round or oval barium collection with folds radiating toward it; if closed, it presents as a filling defect.

5. *Gastric fundus segment.* It is of variable shape and size depending on the habitus of the patient and degree of distention. Neighboring organs (spleen, heart apex, left lobe of the liver) can project over the gastric bubble simulating intrinsic abnormalities.

The pathologic radiology includes:

1. *Achalasia.* The abdominal esophageal segment is narrowed having a smooth conical configuration with preservation of the normal folds. Barium traverses this segment intermittently. In advanced cases, where there is marked dilatation with retention, the examination should be performed after esophageal aspiration and lavage.

2. *Esophageal peptic ulceration.* This is usually secondary to reflux esophagitis and is located in the distal third. If it develops in an island of ectopic gastric mucosa, it can be located anywhere in the esophagus.

3. *Varices.* The varices are usually localized in the distal third, although they have been described elsewhere in the esophagus. Sometimes they can be shown before other signs and symptoms of liver cirrhosis are apparent. They are best demonstrated in the horizontal position and with the esophagus incompletely distended (mucosal study). Depending on their size, 3 roentgenographic patterns can occur as: (a) moderate thickening of folds; (b) small round filling defects; and (c) large cord-like defects. The esophagus might be slightly dilated, because of decreased muscular tone.

4. *Epiphrenic diverticulum.* This is best demonstrated with the patient in the horizontal position.—*Julian B. Alvarez, M.D.*

the vessel and with a tendency to accumulate in rings.

Correlation of these different types of calcification with cerebral physiologic functions reveals that the group with intimal calcifications had a significantly lower cerebral blood flow than the group with medial calcification. The cerebral vascular resistance of the intimal group was greater than that of the medial group, but not significantly so, and there was no significant difference between the group with absence of calcification and the groups with intimal or medial types of calcification.

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Noted was the fact that the lung lesions changed relatively fast (clearing in one site and appearing in another; rapidly developing pneumatoceles with rapid size changes). The pneumatoceles can persist after the patient is clinically cured.—*Julian B. Alvarez, M.D.*

Phibbs, Brendan P., Sundin, Robert E., and Mitchell, Roger S. Silicosis in Wyoming bentonite workers. *Am. Rev. Resp. Dis.*, Jan., 1971, *103*, 1–17. (Address: Dr. Roger S. Mitchell, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver, Colo. 80220.)

"Bentonite" is a term applied to a group of clays mined in Wyoming and in several other parts of this country. These clays, also known as "mintmorrilonite" and "beidelite" are formed from devitrification of volcanic ashes deposited in water and altered in place.

The free silica content of bentonitic clays ranges from 1 to slightly more than 24 per cent. Bentonite is not a true mineral and has, therefore, no precise chemical formula.

Bentonite clays are useful because of the great water-retaining properties of the substance. Common usages include filler substances for crayons, the manufacture of concrete and a lubricant in oil-well drilling.

The mining of bentonite clays is performed in open pits and the dust is usually blown away by air currents. The milling process, however, produces very large quantities of dust indoors.

Dust analysis made in bentonite milling plants revealed dangerous concentrations of silica various times during the past 13 years. Silicotuberculosis developed in 4 patients, and a totally disabling or fatal disease occurred in at least 4 workers after periods of exposure of from 6 to 14 years.

Bentonite dust, once believed to be harmless, must now be added to the list of hazardous dusts because of its content of free crystalline silica.

The roentgenographic findings in the chest of patients developing silicosis due to bentonite exposure are identical to the roentgenographic appearance of silicosis contacted in other forms of exposure.—*John H. Harris, M.D.*

Remy, J. Thymus et myasthenie: aspects radiologiques. (Thymus and myasthenia: radiological aspects.) *Lille méd.*, Nov., 1970, *15*, 1308–1309.

Fifteen per cent of patients with myasthenia have thymomas (lymphoepitheliomas), 80 per cent of which are encapsulated and 35 per cent quite small, down to 5 grams. Many of the tumors are missed in frontal views, and tomography is useless in the absence of gaseous contrast. Pneumomediastinum, however, is likely to outline the tumor wall, and to permit identification of separating planes of tissue although a large tumor by its sheer volume may prevent the migration of air.

Caval phlebography may be of use even without compression of the superior vena cava, since a compressing thymoma may invade the left brachiocephalic trunk without any signs suggesting this complication. Thymic arteriography can be done by injection of subclavian, internal mammary or inferior thyroid vessels, but carries some hazard; preferably it is done bilaterally. Still better is thymic phlebography since the thymic veins unite in 1 or 2 collectors terminating at the inferior border of the left brachiocephalic trunk. This procedure is better tolerated than pneumomediastinum, does not add to respiratory embarrassment, and permits differentiation between tumor and simple hypertrophy.—*Frank A. Riebel, M.D.*

ABDOMEN

MOIR, J. D. Combined duplication of the esophagus and stomach. *J. Canad. A. Radiologists*, Dec., 1970, *21*, 257–262. (From: Saint John General Hospital, Saint John, New Brunswick, Canada.)

The author reports a case in a 9 day old infant who was found to have complete duplication of the esophagus and the stomach.

The case is unusual in that only one esophagus could be filled through the stomach, while the other esophagus was filled retrograde from the stomach following surgery.

An excellent discussion of the complications is given.—*David C. Alftine, M.D.*

HERRERA, CARLOS ALBERTO. Radiología de la regíon esofagocardiotuberositaria normal y patológica. (Normal and pathologic radiology of the esophago-cardio-tuberosity area.) *Rev Argentina Radiol.*, Sept.–Dec., 1970, *33*, 247–262. (Address: Profesor Titular de Radiología de la Facultad de Medicina de Tucumán, Tucumán, Argentina.)

Anatomically this area has been divided into several segments.

1. *Epiphrenic segment.* This segment measures about 3–4 cm.; it is limited inferiorly by the hiatus and superiorly by a primary peristaltic wave provoked by deep inspiration or the Valsalva maneuver. This segment is also known as "the ampulla of Hasse-Strecker," or "the lower functional esophageal sphincter of Lerche."

2. *Intrahiatal segment.* It measures about ½ cm., there being a slight narrowing at this level.

3. *Abdominal segment.* It measures 2–4 cm. Its lateral (left) margin forms with the medial margin of the gastric fundus an acute angle ("the angle of His"). Within the lumen of the stomach, there is a mucosal valve ("the valve of Gubaroff") which corresponds with this external angle. This valve plays an important part in gastroesophageal continence.

4. *Cardiac segment.* This is an oval orifice 2 cm. in longest diameter; in its normal resting state it is closed. A true sphincter has not been demonstrated. It is the diffuse contraction of the abdominal segment that acts as a sphincter.

5. *Major tuberosity (gastric fundus).* This corresponds to the area above a transverse plane through the cardia. The average length is 5 cm.

The normal radiographic appearances are:

1. *Epiphrenic segment.* The ampulla is seen as a transitory smooth oval dilatation appearing during deep inspiration; if this is maintained, the ampulla empties proximally after the peristaltic wave above it subsides.

2. *Intrahiatal segment.* Its projection varies according to position of observation. As seen frontally and in a slight right anterior oblique (RAO) position, it projects above the left diaphragm. In a sharp RAO it projects at or below the diaphragm.

3. *Abdominal segment.* Its length is variable, sometimes it is indistinguishable.

4. *Cardiac segment.* If seen "en face" and opened, it presents as a round or oval barium collection with folds radiating toward it; if closed, it presents as a filling defect.

5. *Gastric fundus segment.* It is of variable shape and size depending on the habitus of the patient and degree of distention. Neighboring organs (spleen, heart apex, left lobe of the liver) can project over the gastric bubble simulating intrinsic abnormalities.

The pathologic radiology includes:

1. *Achalasia.* The abdominal esophageal segment is narrowed having a smooth conical configuration with preservation of the normal folds. Barium traverses this segment intermittently. In advanced cases, where there is marked dilatation with retention, the examination should be performed after esophageal aspiration and lavage.

2. *Esophageal peptic ulceration.* This is usually secondary to reflux esophagitis and is located in the distal third. If it develops in an island of ectopic gastric mucosa, it can be located anywhere in the esophagus.

3. *Varices.* The varices are usually localized in the distal third, although they have been described elsewhere in the esophagus. Sometimes they can be shown before other signs and symptoms of liver cirrhosis are apparent. They are best demonstrated in the horizontal position and with the esophagus incompletely distended (mucosal study). Depending on their size, 3 roentgenographic patterns can occur as: (a) moderate thickening of folds; (b) small round filling defects; and (c) large cord-like defects. The esophagus might be slightly dilated, because of decreased muscular tone.

4. *Epiphrenic diverticulum.* This is best demonstrated with the patient in the horizontal position.—*Julian B. Alvarez, M.D.*

As a consequence of this experience the authors suggest the construction of a routine for diagnostic and therapeutic evaluation.

The patient presented with an elevated serum immunoassayable luteinizing hormone but a clinically negative examination. They suggest that such an early diagnosis of a gonadotropin-producing neoplasm in a man should raise the highly probable diagnosis of a teratocarcinoma of the testes.—*Luther W. Brady, M.D.*

Bluming, Avrun Z., and Templeton, A. C. Histological diagnosis of Burkitt's lymphoma: a cautionary tale. *Brit. M. J.*, Jan., 1971, *1*, 89–90. (From: Makerere University Medical School, Kampala, Uganda.)

In 1969 the World Health Organization published a monograph listing histologic criteria for the diagnosis of Burkitt's lymphoma.

The authors report a patient in whom the histologic differential diagnoses ranged farther afield than other lymphoproliferative or myeloproliferative disorders before being recognized as Burkitt's lymphoma. The case report is presented to illustrate the source of errors that could be encountered in diagnosing the disease. These are difficulties with frozen section, the applicability of imprint preparations, and the technique of first paraffin sections.—*Luther W. Brady, M.D.*

Israel, Harold L., and Goldstein, Robert A. Relation of Kveim-antigen reaction to lymphadenopathy: study of sarcoidosis and other diseases. *New England J. Med.*, Feb., 1971, *284*, 345–349. (From: Sarcoidosis Clinic, Thomas Jefferson University Hospital, Philadelphia, and the Pulmonary Disease Service, Veterans Administration Hospital, Washington, D. C.)

Kveim tests in 37 selected patients with sarcoidosis demonstrated that a reaction could be related to persistent lymphadenopathy and not to duration or activity of illness. Fourteen asymptomatic patients with marked enlargement of the mediastinal and hilar lymph nodes of long duration all reacted strongly.

It has been repeatedly emphasized that the Kveim test is usually positive in early or active sarcoidosis and frequently negative in patients with long standing or inactive disease. Kveim-antigen reactions are most often positive in patients with erythema nodosum, and have been noted more frequently in patients with hilar lymphadenopathy than in patients with pulmonary infiltration.

An experimental study was instituted, applying Kveim tests to patients with known sarcoidosis of 3 types: asymptomatic long standing lymphadenopathy: subacute disease with minimal or no lymphadenopathy; and active granulomatous disease with a normal chest roentgenogram (in 12 patients). The results of this investigation shed new light on the nature of the Kveim reaction, demonstrating a closer relation to lymphadenopathy than to granulomatosis.

Diagnosis of sarcoidosis in each case was supported by histologic evidence of granulomas, negative fungal serologic tests and tuberculin tests. The cases were classified according to duration and activity of illness and the appearance of the roentgenogram of the chest according to standard usage.

Records were reviewed of 55 other patients tested for diagnostic purposes. The patients were classified according to the appearance of the chest roentgenogram.

Positive reactions were obtained in only 3 of 12 patients with subacute active sarcoidosis.

Patients with Hodgkin's disease and histoplasmosis had negative Kveim tests.

Positive Kveim tests in this series were obtained in 23 (88 per cent) of 26 patients with Stage I disease, in 16 (70 per cent) of 23 with Stage II disease, and in 3 (50 per cent) of those with Stage III disease.

Of special interest were the 11 patients with active cutaneous or hepatic granulomas whose chest roentgenograms appeared normal. Kveim tests were negative in 10 cases, including 2 others with splenomegaly.

A retrospective analysis of 55 patients tested for diagnostic purposes showed the highest percentage of positive reactions in those with Stage I disease (hilar or mediastinal lymphadenopathy alone), followed by Stage II (lymphadenopathy and infiltration) and Stage III (pulmonary infiltration alone). Patients with Stage I and II disease and negative Kveim tests had hilar or mediastinal lymph node enlargement that, as a rule, disappeared in 6 months or less (those with positive reactions had hilar changes that remained for longer intervals). It is *evident that persistent lymphadenopathy is essential to the Kveim-antigen reaction.*

The high frequency of positive tests in "early" sarcoidosis reflects the fact that most cases of sarcoidosis begin with hilar lymphadenopathy. Cases described here demonstrate that the duration of sarcoidosis cannot be accurately judged from roentgenographic characteristics.

Kveim-test responsiveness disappears when lymphadenopathy is gone. Kveim-antigen reaction represents a sluggish immunologic response, intimately related to lymph node involvement, that fits none of the usual categories of allergic hypersensitivity.

If evidence accumulates that the response is not restricted to patients with sarcoidosis, the concept that Kveim antigen must be prepared from lymph nodes or spleens involved by sarcoidosis must be re-examined. Extracts prepared from granulomatous lungs and granuloma-free lymph nodes or spleens should be tested.—*Stephen N. Tager, M.D.*

Kawasaki, Hiroshi, Imasato, Katsujiro, Kodama, Takeshi, Kimoto, Eiji, Nagaoka, Shoji, and Tanaka, Yasuhisa. Immunofluorescent staining of the gastric mucosa-specific epithelial glycoprotein in metastasized gastric cancer cells: a case report of Krükenberg tumor. *Kurume M. J.*, 1970, *17*, 153–160. (From: Departments of Pathology, Clinical Pathology, and Internal Medicine, Kurume University School of Medicine, Kurume, Japan.)

Previous immunofluorescent studies revealed that the gastric mucosa-specific epithelial glycoprotein appeared in mucoid cells and secreted mucus in the normal gastric mucosa, as well as even in the diffusely and invasively proliferated gastric cancer cells in the mucosal, submucosal and muscular layers.

Krükenberg tumor, which metastasizes frequently not only to the ovary but also to various other organs, is generally characterized by mucin-producing epithelial cells having a signet ring form. The primary tumor is recognized to be most often in the gastrointestinal tract.

The case report is that of a 35 year old woman with a scirrhous type carcinoma involving the stomach and duodenal region. The ascites was bloody and there was a Class v Papanicolaou smear. Considered inoperable she received only palliative treatment and expired in a few months.

At postmortem examination there was extensive tumor involvement of the lungs, and virtually every organ in the peritoneal cavity, including the ovaries. Microscopically signet ring cells with considerable mucoid substance were seen in all sections made from the involved multiple organs. The tumor was classified as an adenocarcinoma of tubular and mucocellulo-nodular type. This was further diagnosed as a Krükenberg tumor with a primary gastric cancer.

This impression was confirmed by preparing rabbit antisera against the epithelial glycoprotein (blood-group active) of human gastric mucosa and labeling with fluorescein isothiocynate. The immunofluorescent tissue sections of the primary gastric and various metastasized organs demonstrated intensely fluoresced cancer cells especially of signet ring form, correlating well with PAS staining. It might therefore be deduced that such metastasized cancer cells in various organs apart from the primary gastric region persisted in producing or secreting the epithelial glycoprotein specific for the gastric mucosa. Such a fluorescent antibody technique might be of great aid in determining the primary lesion in some types of cancerous metastases.—*Donald M. Monson, M.D.*

RADIOISOTOPES

Rollo, F. David. ^{125}I for measuring the radioiodine uptake of the thyroid. *J. Nuclear Med.*, Jan., 1971, *12*, 8–13. (From: Upstate Medical Center, Syracuse, N. Y.)

A technique for measuring the I^{125} in thyroid uptake is described which is based on the 2 distance (2D) determination method. This approach requires one additional measurement besides the standard ORINS technique.

The author states that I^{125} uptake with the 2D method has the following advantages: (a) reducing the dose but improving counting statistics; (b) removing certain inaccuracies of the conventional method; (c) reducing radiation to the thyroid; (d) affording high counting efficiency; and (e) permitting long term evaluation of the biologic retention of the iodine in the thyroid.—*Abbas M. Rejali, M.D.*

Hoffer, Paul B., Beck, Robert N., Lembares, Nicholas, Charleston, Donald B., and Gottschalk, Alexander. Use of lithium-drifted germanium detectors for clinical radionuclide scanning. *J. Nuclear Med.*, Jan., 1971, *12*, 25–27. (From: University of Chicago and the Argonne Cancer Research Hospital, Chicago, Ill.)

In this communication, incorporation of a lithium-drifted germanium detector into a rectilinear scanner is evaluated for its usefulness in radioisotope scanning. Its potential advantages are discussed with particular emphasis on the reduction of scatter through its inherently high resolution.

It is also mentioned that the chief disadvantages of the lithium-drifted germanium detectors are lack of availability of large crystals, low sensitivity, and the requirement that this detector be operated at liquid nitrogen temperature.—*Abbas M. Rejali, M.D.*

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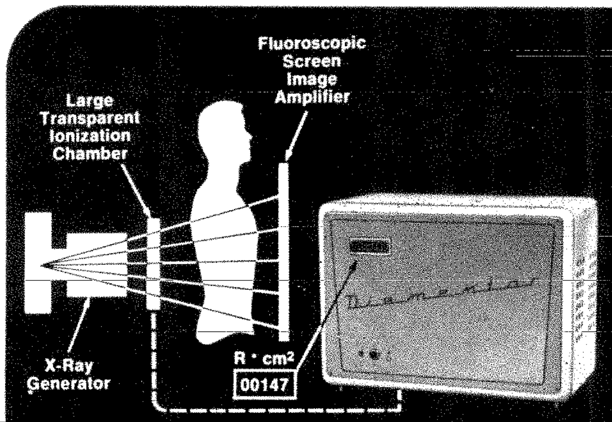
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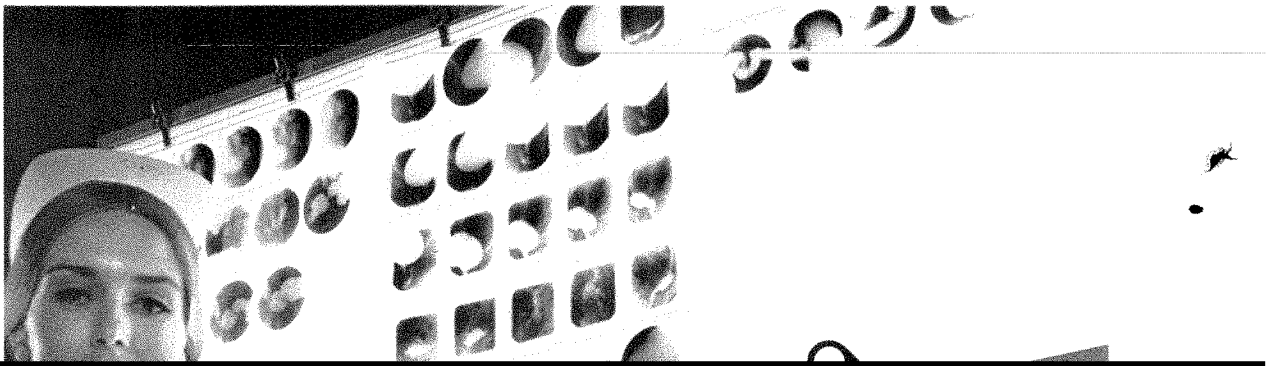

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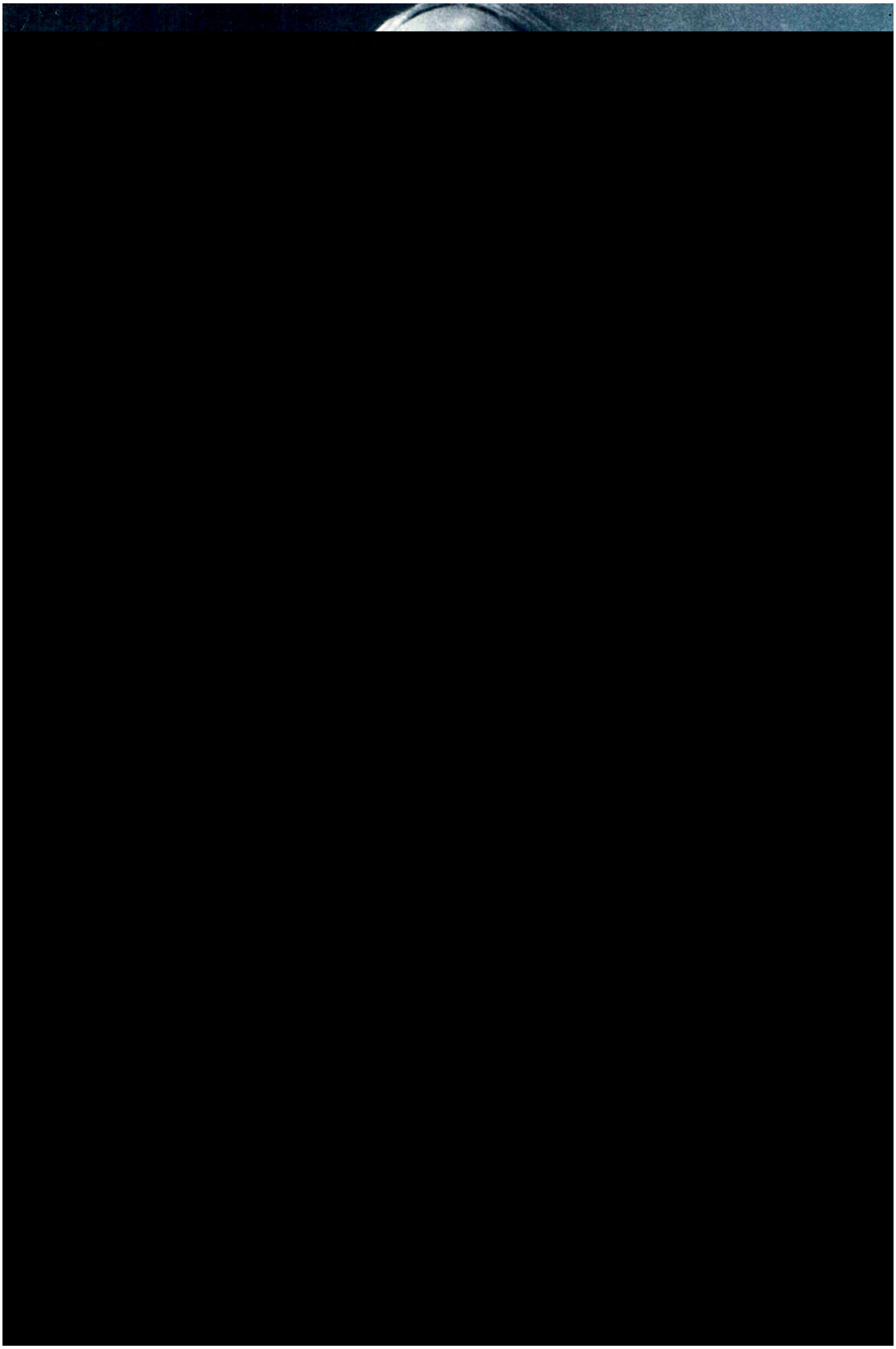

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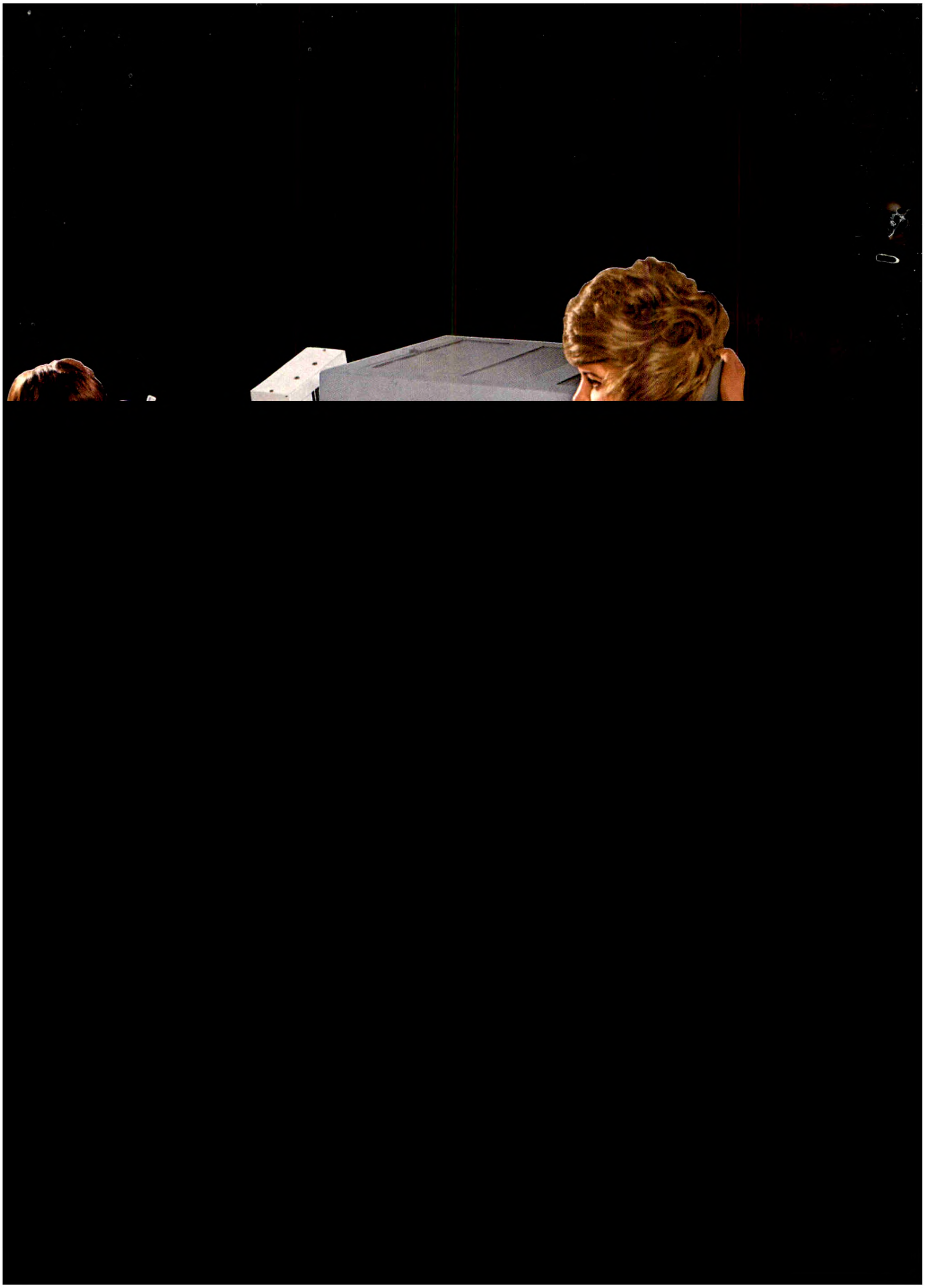

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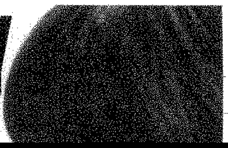

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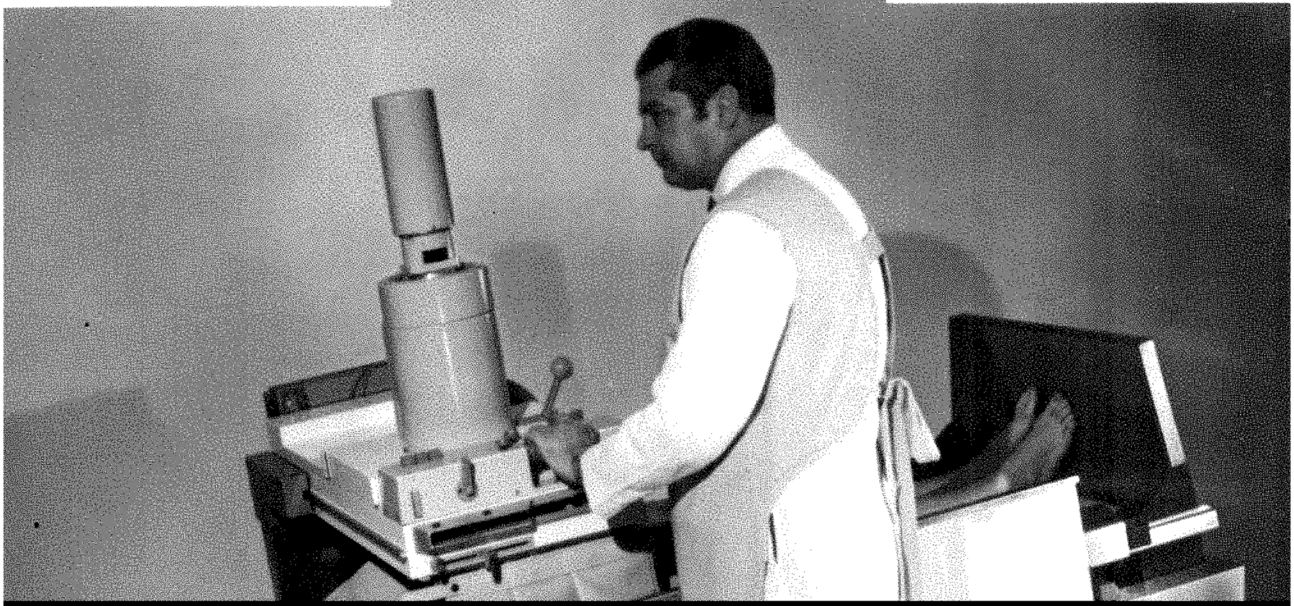

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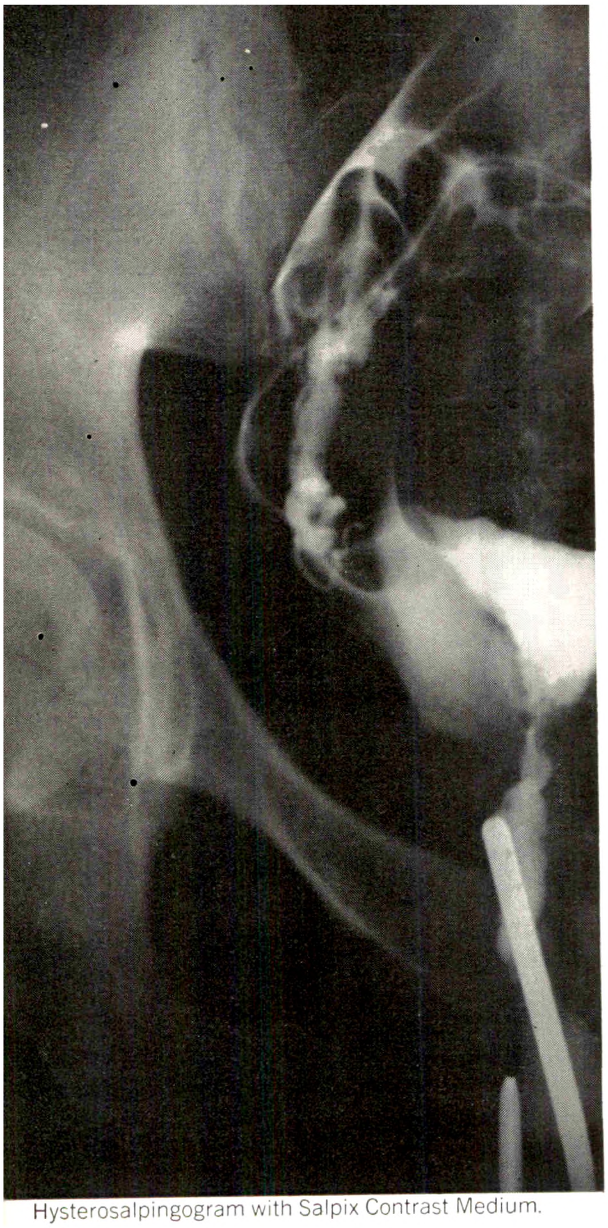

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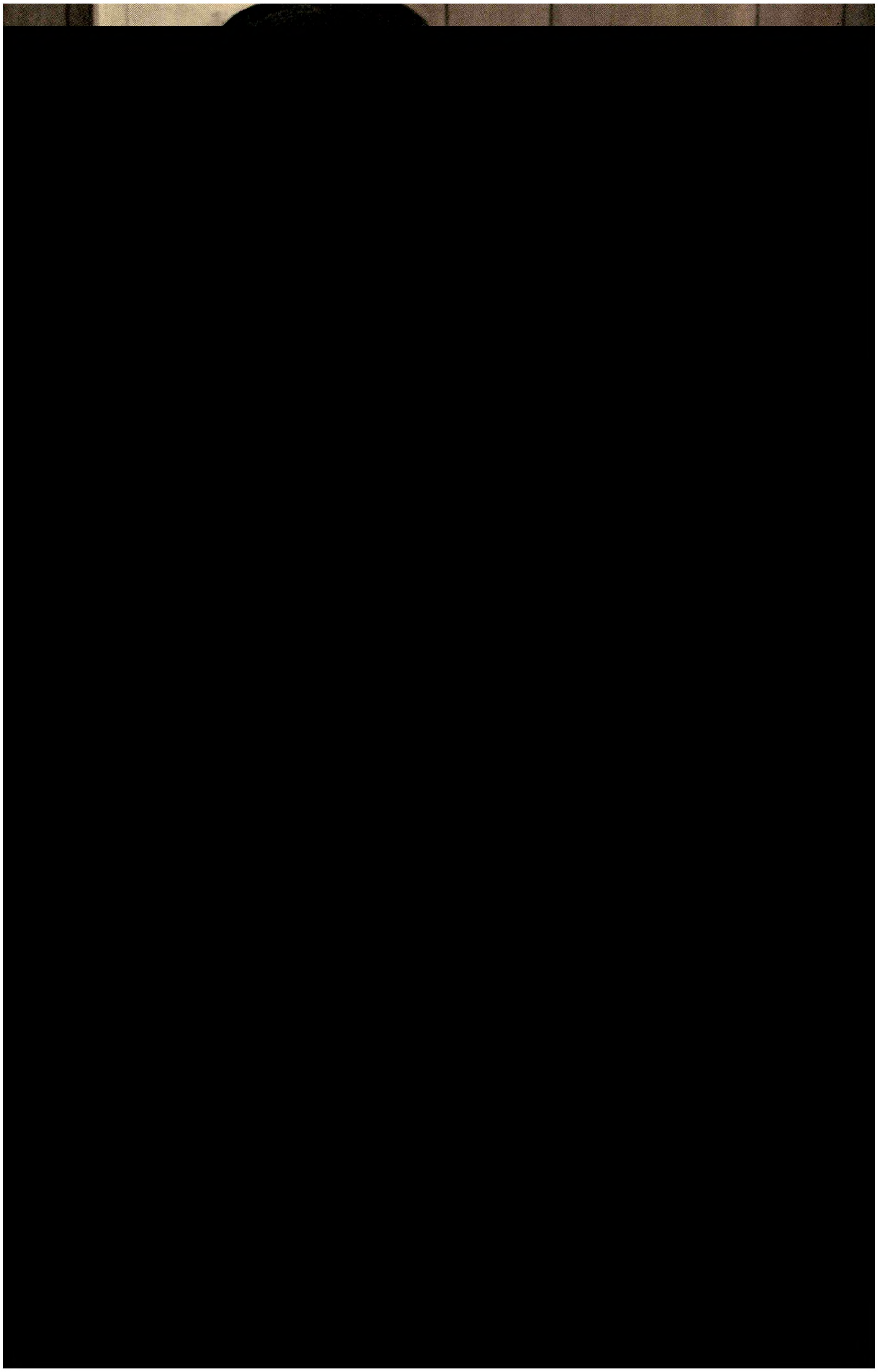

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